

Health risks of ozone



from long-range transboundary air pollution



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Abstract

Ozone is a highly oxidative compound formed in the lower atmosphere from gases (originating to a large extent from anthropogenic sources) by photochemistry driven by solar radiation. Owing to its highly reactive chemical properties, ozone is harmful to vegetation, materials and human health. In the troposphere, ozone is also an efficient greenhouse gas. This report summarizes the results of a multidisciplinary analysis aiming to assess the effects of ozone on health. The analysis indicates that ozone pollution affects the health of most of the populations of Europe, leading to a wide range of health problems. The effects include some 21 000 premature deaths annually in 25 European Union countries on and after days with high ozone levels. Current policies are insufficient to significantly reduce ozone levels in Europe and their impact in the next decade.

Keywords

AIR POLLUTANTS, ENVIRONMENTAL - adverse effects
OZONE
RISK ASSESSMENT
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EUROPE

Health risks of ozone from long-range transboundary air pollution

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Convention on Long-range Transboundary Air Pollution

Contents

Abbreviations	vii
Executive summary	ix
1. Introduction	1
Structure of the report	2
2. Hazard assessment of ozone	3
Ozone toxicokinetics	3
Acute responses	5
Chronic effects in humans	11
Thresholds	15
Susceptible groups	17
Health implications	18
3. Sources of ozone precursors	21
Ozone formation and atmospheric transport	21
Sources of ozone precursor emissions	23
Projections of future emissions of ozone precursors	24
Interactions of ozone precursor emissions with other environmental problems	30
4. Ozone levels	31
Monitoring results	31
Observed ozone trends	39
Modelling techniques and validation	47
Ozone and climate change	50

5. Population exposure projections	53
Exposure versus ambient measurements	54
Population exposure modelling	56
6. Risk estimates	59
Effects of ozone on mortality	60
Estimation of impacts on morbidity	65
Health effects and cost–benefit analysis	71
Transferability between populations	72
Evidence on reversibility of the health impacts	73
7. Conclusions	75
Ozone air pollution is a significant health hazard in Europe	75
Health effects of long-range transboundary ozone are most likely proportional to the contribution of long-range sources to ozone exposure levels	75
Current policies lead to only a small reduction in risk	76
Key uncertainties, research needs and policy implications	76
References	79
Annex 1. List of Working Group members and reviewers	95

Abbreviations

Organizations and programmes

ACS	American Cancer Society
AHSMOG	Adventist Health and Smog (study)
AirBase	European air quality database (http://air-climate.eionet.europa.eu/databases/airbase/)
CAFE	Clean Air for Europe Programme of the European Commission (http://europa.eu/scadplus/leg/en/lvb/l28026.htm)
CAFE CBA	CAFE Cost–Benefit Analysis project (http://www.cafe-cba.org)
COMEAP	Committee on the Medical Effects of Air Pollution
ECHRS	European Community Respiratory Health Survey
EEA	European Environment Agency (www.eea.europa.eu)
EMEP	Cooperative Programme for Monitoring and Evaluation of the Long-range Transmission of Air Pollutants in Europe (www.emep.int)
EU	European Union
EU15	European Union with 15 Member States as it existed between 1995 and 2004
EU25	European Union with 25 Member States as it existed between 2004 and 2007
GAINS	Greenhouse gas – Air pollution INteractions and Synergies
IPCC	Intergovernmental Panel on Climate Change
ISAAC	International Study of Asthma and Allergies in Childhood
MSC-W	EMEP Meteorological Synthesizing Centre – West, hosted by the Norwegian Meteorological Institute
RAINS	Regional Air Pollution Information and Simulation model of IIASA

UNECE	United Nations Economic Commission for Europe (www.unece.org)
IIASA	International Institute for Applied Systems Analysis, Vienna (http://www.iiasa.ac.at/)
OECD	Organisation for Economic Co-operation and Development

Technical terms

AOT40/AOT60	accumulated ozone above the level of 40/60 ppb, a measure of cumulative annual ozone concentrations used as indicator of vegetation (health) hazards
CI	confidence interval (a measure of statistical uncertainty in numerical estimates)
CIMT	carotil intima-media thickness
CLE	current legislation (concerning emission of pollutants to the atmosphere)
COPD	chronic obstructive pulmonary disease
DALY	disability-adjusted life-year (a measure of health burden)
ELF	epithelial lining fluid
FEF ₂₅₋₇₅	forced expiratory flow between 25% and 75% FVC
FEF ₇₅	forced expiratory flow at 75% FVC
FEV ₁	forced expiratory volume in 1 second (measure of respiratory function)
F gases	fluorinated greenhouse gases (hydrofluorocarbons, perfluorocarbons and sulfur hexafluoride)
FVC	forced vital capacity (measure of respiratory function)
HIS	United States Health Interview Study
HRV	heart rate variability

ICD-9	International Statistical Classification of Diseases and Related Health Problems, ninth revision
LRS	lower respiratory symptoms
LRTAP	long-range transboundary air pollution
MTFR	maximum technologically feasible reduction (concerning emission of pollutants to the atmosphere)
NO _x	nitrogen oxides
NO _y	reactive nitrogen oxide
OH	hydroxyl radical
OFIS	ozone fine structure model
PAN	peroxyacetyl nitrate
PM	particulate matter
ppb/ppm	parts per billion/parts per million (volumetric unit of concentration)
PPP\$	purchasing power parity dollars
RADs	restricted activity days
RHAs	respiratory hospital admissions
SOMO35	sum of maximum 8-hour ozone levels over 35 ppb (70 µg/m ³) (a measure of accumulated annual ozone concentrations used as an indicator of health hazards (overall long-term ozone levels); see Box 4.2 (page 36) for a more complete definition)
SOMO0	sum of maximum 8-hour ozone levels without a threshold
VOC	volatile organic compound

Executive summary

This report summarizes the results of a multidisciplinary analysis aiming to assess the health effects of ozone, and especially the part that is contributed by remote sources. The analysis indicates that ozone pollution affects the health of most of the populations of Europe, leading to a wide range of health problems. Currently implemented policies are not sufficient to reduce impacts significantly in the next decade.

Ozone is a highly oxidative compound formed in the lower atmosphere from gases (originating to a large extent from anthropogenic sources) by photochemistry driven by solar radiation. Owing to its highly reactive chemical properties, ozone is harmful to vegetation, materials and human health. In the troposphere, ozone is also an efficient greenhouse gas.

Health hazard

As to short-term exposures, recent epidemiological studies have strengthened the evidence that daily exposures to ozone increase mortality and respiratory morbidity rates. These studies have provided information on concentration–response relationships and effect modification. In short-term studies on pulmonary function, lung inflammation, lung permeability, respiratory symptoms, increased medication usage, morbidity and mortality, ozone appears to have effects independent of other air pollutants such as particulate matter (PM). This notion that ozone may act independently is strengthened by controlled human studies and experimental animal studies showing the potential of ozone per se to cause adverse health effects, especially in vulnerable people. Controlled human studies on PM and ozone combined corroborate this view.

As to long-term exposures, new epidemiological evidence and experimental animal studies on inflammatory responses, lung damage and persistent structural airway and lung tissue changes early in life also indicate effects of long-term exposure to ozone. This evidence is still too limited for firm conclusions to be drawn, however, but in the future it may be possible to identify health effects from long-term exposure to ozone.

Sources and emission trends

The most important pollutants that play a role in the formation of tropospheric ozone include nitrogen oxides (NO_x) and volatile organic compounds (VOCs) as well as, to a lesser but still significant extent, methane and carbon monoxide. The pace of photochemical reactions forming ozone in the atmosphere depends on

solar radiation and temperature. Inside and close to urban areas, ozone concentrations may be depressed because of reactions with NO_x but further downwind (in rural areas) both NO_x and VOCs tend to promote ozone formation.

Emissions of NO_x occur in the most densely populated areas, particularly in northwestern Europe. On the other hand, VOC emissions are more evenly distributed in Europe, the main anthropogenic sources being traffic and solvent use. In European Union (EU) countries, emissions of ozone precursors are expected to decline further, even assuming accelerated economic growth, dropping by 2020 to half the 2000 levels. For these pollutants, contributions from the traditionally dominant source sectors (energy production, industry and road transport) will significantly decrease. In the future, the relative roles of other sectors that currently have less strict legislation (including shipping, diesel-powered heavy-duty and off-road vehicles for NO_x and solvent use for VOC) will increase. However, the lack of relevant, stringent legislation in many non-EU countries may result in further increases in ozone precursor emissions in these parts of the region covered by the Convention on Long-range Transboundary Air Pollution.

Ozone levels and trends

Even though emissions of ozone precursors have fallen over large parts of Europe since the late 1980s, ozone levels continue to cause health concerns, with the highest levels in south and central Europe. Concentrations in southern Europe are higher than in northern Europe and are higher in rural than in urban areas. Peak ozone values fell in several regions in Europe during the 1990s, while there was no trend in the sum of maximum 8-hour ozone levels over 35 ppb ($70 \mu\text{g}/\text{m}^3$) (SOMO35), a metric used for ozone health impact assessment. Ozone levels are strongly influenced by annual variations in weather conditions and trends in the hemispheric background concentrations.

Simulations of SOMO35 for 2010 indicate that emissions overall will be slightly lower than in 2000 in central Europe. However, in some (urban) areas, the combination of reduced NO_x titration and an increasing contribution of hemispheric background ozone is leading to increasing ozone levels in cities and increased population exposures to ground-level ozone. Regional differences in ozone levels across Europe are expected to decrease in the next decade. Exposures in continental Europe are projected to go down by 20–30% in northern Italy, Germany, southern France and Switzerland, and to rise in Scandinavia and the British Isles.

Human exposure to ozone during the winter is reduced because more time is spent indoors. Building structures and slow rates of ventilation reduce ozone penetration indoors even during the summer.

Health impact estimates

It is estimated that some 21 000 premature deaths per year are associated with ozone exceeding $70 \mu\text{g}/\text{m}^3$ measured as a maximum daily 8-hour average in 25 EU countries (EU25). The slight decline in ground-level ozone expected to result from current legislation, and taking account of current policies addressing climate change (the CLE scenario), is estimated to reduce premature mortality by only some 600 cases per year between 2000 and 2020. Markedly larger (around 40%) reductions could be achieved by implementing the maximum technically feasible reduction (MTFR) scenario.

Ozone is also associated with 14 000 respiratory hospital admissions annually in EU25. It affects the daily health of large populations in terms of minor restricted activity days, respiratory medication use (especially in children), and cough and lower respiratory symptoms. The estimated figures are between 8 million and 108 million person-days annually, depending on the morbidity outcome. Expected reductions in morbidity outcomes related to the implementation of current policies (CLE scenario) are more significant than those for mortality, ranging from approximately 8% (respiratory medication use of adults) to 40% (cough and lower respiratory symptoms in children). Nevertheless, hospital admissions associated with ozone exposure are expected to increase owing to changes in population structure and larger populations of older people at risk. The current health impact estimates consider only acute health effects, and do not account for possible effects at short-term ozone exposure levels below $70 \mu\text{g}/\text{m}^3$ or possible effects from long-term exposures.

While the premature mortality associated with ozone in EU25 is substantially lower than that associated with fine PM, ozone is nevertheless one of the most important air pollutants associated with health in Europe.

1. Introduction

In most countries of the United Nations Economic Commission for Europe (UNECE) region, ambient air quality has improved considerably in the last few decades. This improvement was achieved by a suite of measures to reduce harmful air emissions, including those stipulated by the various protocols under the Convention on Long-range Transboundary Air Pollution (LRTAP). On the other hand, there is convincing evidence that current levels of air pollution still pose a considerable risk to the environment and human health.

The Convention on LRTAP has been extended by eight protocols. The Protocol to Abate Acidification, Eutrophication and Ground-level Ozone was adopted by the Executive Body for the Convention in Gothenburg, Sweden in November 1999, has been signed by 23 Parties, and entered into force on 17 May 2005. While early agreements on LRTAP were driven by concerns about the transboundary transport of acidifying pollutants, effects on human health have attracted more and more attention in recent years. These concerns led to the creation of the Joint WHO/Convention Task Force on the Health Aspects of Air Pollution. The main objective of this Task Force, which is chaired by WHO, is to prepare state-of-the-art reports on the direct and indirect effects of air pollutants on human health.

WHO was already collaborating with the Convention on assessing the health effects of ozone before the Task Force was created. A joint workshop was organized by the Convention, the WHO European Centre for Environment and Health and the MRC Institute for Environment and Health and was hosted by the United Kingdom Department of the Environment in Eastbourne on 10–12 June 1996. The workshop formulated recommendations related to the information and methods needed to improve assessments of the health impacts of ozone in Europe (1).

This report provides a concise summary of current knowledge on the risks to health of ozone from LRTAP. It is targeted at various groups supporting implementation of the Convention, including the Working Group on Strategies and Review and the Executive Body. The report is also directed at decision-makers at national level concerned with pollution abatement policies, as well as to scientists who can contribute further information on all stages of assessing the risks to health posed by ozone.

The main aim of this report is to provide a scientific rationale for estimating the magnitude, spatial distribution and trends in the health burden caused by exposure to ozone in ambient air in Europe, and in particular the contribution to ozone levels from the long-range transport of pollutants. It combines the evi-

dence generated in the recent update of WHO's air quality guidelines (2) and in the work on modelling and assessment of ozone levels conducted for the Convention as well as for the European Commission's Clean Air for Europe (CAFE) programme.

This report focuses on tropospheric ozone, which, owing to its highly oxidative properties, is harmful to human health, vegetation and materials. In the upper layer of the atmosphere, the stratosphere, ozone is formed from oxygen in reactions initiated by solar radiation. Approximately 90% of atmospheric ozone is formed in the stratosphere, where it plays a highly valuable role in absorbing ultraviolet light, an excess of which is harmful to life on Earth.

The long-range contribution to ozone levels is equivalent to the regional background of ozone, which includes naturally occurring ozone. This contribution is not strongly influenced by single emission sources and should be roughly equivalent to (a) those measured at rural (background) locations and (b) air pollution levels estimated by regional air transport models.

Structure of the report

Following a concise summary on hazard identification given in Chapter 2, largely based on the results of the recent update of WHO's air quality guidelines (2), Chapter 3 provides an overview of the sources of ozone precursors (*pressure*). The emission data are derived both from national submissions to the UNECE secretariat and from expert estimates. Atmospheric distribution and transformations, as well as current ambient levels and trends of tropospheric ozone (*state*), are described in Chapter 4. A section on ozone levels from ambient air monitoring is complemented by a description of estimates from the Unified Eulerian EMEP model. These data are a prerequisite for Chapter 5 on exposure assessment.

The overall assessment of the health effects is completed in Chapter 6 using a risk assessment approach, integrating the information on exposure, concentration–response and background frequency of the considered effect. Most of the calculations were made in support of the CAFE programme, following the methodology agreed by the Joint WHO/Convention Task Force in 2004.

The first draft of this report was prepared in 2005. Its consecutive drafts were discussed by the Task Force at meetings in 2006 and 2007, providing input to the discussions of the Working Group on Effects. The revised and updated draft was on the agenda of the 11th meeting of the Task Force, held in Bonn on 17–18 April 2008, which formulated the “key messages” of each chapter, executive summary and conclusions of the assessment presented in Chapter 7. The executive summary was used as a contribution to the report of the Task Force presented to the 27th session of the Working Group on Effects (3).

2. Hazard assessment of ozone

KEY MESSAGES

- Ozone is a highly reactive gas that triggers oxidative stress when it enters the airways.
- Adverse structural, functional and biochemical alterations in the respiratory tract occur at current ozone levels, as confirmed by animal and autopsy data.
- Exposure to ozone increases daily mortality and morbidity levels in populations.
- The risk of effects increases in proportion to the ozone level, with a significant increase in mortality observed above 50–70 $\mu\text{g}/\text{m}^3$ (measured as a 1- or 8- hour average).
- Evidence of chronic effects is currently less conclusive. New evidence of such effects is emerging, such as that on small airway function and possibly on asthma development; if these are confirmed, the health concerns will increase.

Evaluation of the accumulated evidence on the hazards of exposure to ozone was recently completed by WHO in the 2005 update of its air quality guidelines (2). This chapter is based, to a large extent, on the results of this evaluation.

Ozone toxicokinetics

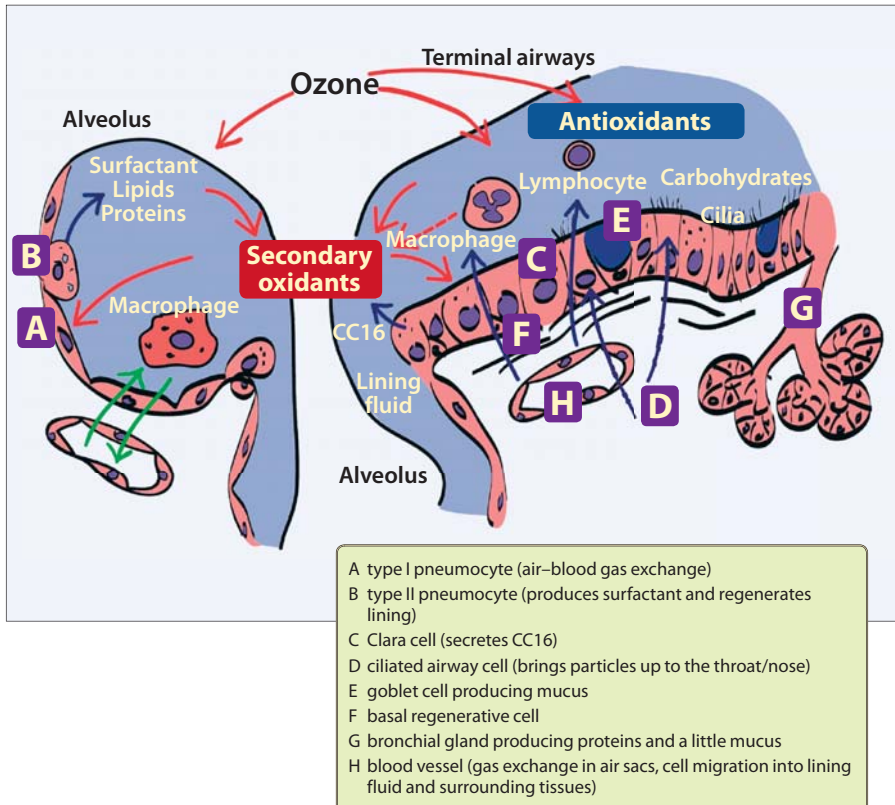
Because of its high reactivity and low solubility in water, exposure to ozone via liquid or solid media is negligible and ozone uptake is thus almost exclusively by inhalation. As to other routes of exposure, there is evidence of effects in the tear duct epithelial cells of individuals exposed to ambient ozone levels (4) and in the skin of laboratory animals exposed to extremely high concentrations (5). Nevertheless, it is likely that ozone effects on skin are restricted to the upper layers of the dermis and that no absorption occurs in its innermost compartments. Thiele et al. (6) demonstrated that short-term exposure of mice to high levels of ozone significantly depleted vitamins C and E and induced malondialdehyde formation in the upper epidermis but not in underlying layers. There is currently no evidence that oxidative stress by ambient ozone levels would interfere with epidermal integrity and barrier function and predispose to skin diseases.

Most absorption of ambient ozone occurs in the upper respiratory tract and conducting intrathoracic airways (7,8). Total ozone uptake is at least 75% in adult males (9). The rate of absorption may change, being inversely proportional to flow rate and increasing as tidal volume increases (8). As tidal volume increases, there is a shift from nasal to oral breathing, with most of the inhaled air entering

through the mouth at flow rates exceeding 40 litres per minute (10). Since ozone removal in the upper respiratory tract is lower for oral than for nasal inhalation, ozone penetration into the lungs is much higher in people engaged in vigorous physical activity. Age and gender also influence ozone absorption, in both quantity and topography, because of variations in airway size and the tissue surface of the conducting airways, leading to higher levels of absorption in children and women (7).

Diffusion of ozone across the airway epithelial lining fluid (ELF) (Fig. 2.1) is determined by its reactivity, and direct contact of ozone with airway epithelium seems to be small (11). ELF contains substrates such as ascorbic acid, uric acid, glutathione, proteins and unsaturated lipids that may undergo oxidation mediated by ozone (12), thus preventing (or minimizing) damage to the underlying epithelium. ELF is constantly renewed by the mechanical input provided by the coordinated movement of airway ciliated cells, producing new biological substrates to react with ozone and thus acting as a chemical barrier against this pollutant. However, oxidation of some components of ELF may generate bioactive compounds, such as lipid hydroperoxides, cholesterol ozonization products,

Fig. 2.1. Interactions of ozone with the terminal airway lining fluid and cells



ozonides and aldehydes, with the potential to elicit inflammation and cell damage (13).

Modelling studies show that the total percentage ozone taken up by the lungs is not markedly affected by age, but this changes when the amount of ozone absorbed is normalized by the regional surface area of the different segments of the respiratory tract (8). Malnutrition may interfere with the availability of antioxidant substances in ELF, such as vitamin E. Pre-existing pulmonary disease, such as chronic bronchitis, asthma or emphysema, leads to mechanical unevenness of airflow because of regional differences in the time constants of parallel respiratory units, thus interfering with the tissue dosimetry of ozone. Thus, for any given ambient level of ozone concentration, its toxicity, preferential site of damage and pathogenetic mechanisms may vary depending on various factors in the human receptor.

Studies on rodents and non-human primates to relatively high levels of ozone have shown structural changes in the peripheral parts of the lung. The structural changes in the primates were found after six months of exposure to 0.5 ppm ozone. The age at which the monkeys were exposed corresponds approximately to early childhood in humans: the first 2–3 years of life. After discontinuation of exposure the animals were followed for another six months (equivalent to about 1½ years for humans) but the changes persisted. Though not directly relatable to a disease, persisting structural changes should be regarded as an adverse effect (14,15).

Acute responses

For acute responses (other than the newly emerging area of cardiovascular function), there is a very large and rapidly growing literature that was summarized as part of the WHO air quality guidelines development process (3). Judgement is required in the interpretation of pulmonary system effects, in that some of the measurable effects may not be worthy to be considered adverse. By contrast, any excess hospital admissions and excess daily mortality attributable to ozone is clearly adverse.

Epidemiological studies used daily ozone levels (measured as maximum daily 1- or 8-hour average) as the exposure indicator. Recently published meta-analyses use the daily average to ensure the comparability of the results of various studies. All three indices are highly correlated. The widely used conversion of 1-hour maximum, 8-hour maximum and daily average is 20:15:8 (16). The WHO air quality guidelines (2) refer to the 8-hour average, as being more closely related to the average daily exposure and inhaled dose.

Pulmonary system effects

Very many experimental studies have been performed on the acute effects of ozone exposure in humans. They have employed various approaches: controlled

exposures at rest or during exercise; single or continuous exposures; exposures at ambient levels; and evaluating the effects of ozone on subjects with pre-existing pulmonary disease such as asthma or chronic bronchitis. The studies listed in the WHO air quality guidelines (Annex 1, Table 1) (2), on the acute effects of ozone exposure on physiological parameters in humans, support the following conclusions.

- There is solid evidence that short-term exposure to ozone impairs pulmonary function.
- Controlled exposures indicate that transient obstructive pulmonary alterations may occur for 6.6-hour exposures at an ozone level of $160 \mu\text{g}/\text{m}^3$, a concentration frequently surpassed in many locations in the world.
- People with asthma and allergic rhinitis are somewhat more susceptible to transient alterations in respiratory function caused by acute exposure to ozone.
- Changes in pulmonary function and depletion of airway antioxidant defences are immediate consequences of ozone exposure. Increase in inflammatory mediators, upregulation of adhesion molecules and inflammatory cell recruitment can be detected hours after exposure and may persist for days.
- Ozone enhances airway responsiveness in both healthy individuals and asthmatics.
- Studies conducted under field conditions, such as summer camps, have detected transient functional effects at ozone levels considerably lower than those observed in controlled exposures. Various factors may account for this discrepancy: concomitant exposure to other pollutants (including other components of the photochemical smog) and difficulties in precisely determining individual exposure (present and past). On the other hand, one has to consider that the lower threshold for adverse effects may be influenced by the higher number of days of observation in such studies, thus increasing the power of detecting a significant effect.

The vast majority of the epidemiological studies considered in the 2005 global update of the guidelines (2) obtained positive and significant associations between variations in ambient ozone levels and increased morbidity. School absenteeism, hospital admissions or emergency department visits for asthma, respiratory tract infections and exacerbation of existing airway disease were the most common health end-points. The effects were manifested among children, elderly people, asthmatics and those with chronic obstructive pulmonary disease (COPD). The magnitude of the risk for respiratory morbidity associated to an increase of $20 \mu\text{g}/\text{m}^3$ ozone ranged from zero to 5%. The estimated magnitude of the increase in risk found by various studies is presented in more detail in Chapter 6.

Exposure to ozone has been shown to increase the likelihood of wheeze and chest tightness, increase the risk of morning symptoms of asthma, and reduce

morning peak expiratory flow rates (17,18) in children with lower birth weights or those born prematurely.

There are large multi-city studies relating the numbers of hospital admissions for respiratory diseases (19) and COPD (20) to ambient ozone levels. Such associations were robust enough to persist after controlling for temporal trends in admission rates, day-of-the-week and seasonal effects, gaseous and particulate air pollution, and climatic factors. Effects of ozone on respiratory admissions seem stronger during warmer weather. A meta-analysis by WHO of the European studies (21) provided summary risk estimates for respiratory admissions in the age ranges 15–64 and ≥ 65 years of 1.001 and 1.005 per 10 $\mu\text{g}/\text{m}^3$ ozone, respectively. However, the variability of the results was large and the lower limit of 95% confidence interval (CI) was below 1 (Table 2.1). Three estimates were available for respiratory admissions in children aged 0–14 years; a meta-analysis of these estimates gave a summary relative risk of 0.999 (21).

Cardiovascular system effects

The effect of ambient air pollution on cardiovascular function and the initiation and progression of cardiovascular disease in laboratory animals and human

Table 2.1. Relative risk estimates and confidence intervals for a 10- $\mu\text{g}/\text{m}^3$ increase in daily, maximum 1-hour or maximum 8-hour average ozone for all-cause and cause-specific mortality and respiratory hospital admissions in recent meta-analyses

Meta-analysis/ outcome/disease	Age group (years)	Relative risk (95% CI)	Number of studies analysed
<i>Bell et al. (16)^a</i>			
All-cause mortality, all seasons	All ages	1.004 (1.003–1.009)	32
All-cause mortality, summer	All ages	1.007 (1.004–1.011)	10
Cardiovascular mortality, all seasons	All ages	1.005 (1.003–1.008)	18
Cardiovascular mortality, summer	All ages	1.012 (1.004–1.020)	4
<i>Ito et al. (31)^b</i>			
All-cause mortality	All ages	1.002 (1.001–1.003)	43
<i>Levy et al. (32)^b</i>			
All-cause mortality	All ages	1.002 (1.002–1.003)	46
<i>WHO (33)^c</i>			
All-cause mortality	All ages	1.003 (1.001–1.004)	15
Respiratory mortality	All ages	1.000 (0.996–1.005)	12
Cardiovascular mortality	All ages	1.004 (1.003–1.005)	13
Respiratory hospital admissions	0–14	Not observed	3
	15–64	1.001 (0.991–1.012)	5
	≥ 65	1.005 (0.998–1.012)	5

^a Daily. ^b Maximum 1-hour. ^c Maximum 8-hour.

populations is an emerging field of interest and one of intense study. In studies of acute responses in humans, however, there are difficulties in separating the effects due to peaks in particulate matter (PM) concentrations from those that may be due to ozone.

Park et al. (22) conducted a study on 603 men in the Boston, Massachusetts area who were enrolled in the Veterans Administration Normative Aging Study and were undergoing routine electrocardiographic monitoring, including measurement of heart rate variability (HRV). Reduced HRV is a well-documented risk factor for cardiac disease. Low-frequency HRV was reduced by 11.5% (95% CI 0.4–21.3) per 2.6- $\mu\text{g}/\text{m}^3$ increment in the previous 4-hour average of ozone, and the effect was stronger in men with ischemic heart disease and hypertension. There were also significant associations of HRV with PM_{10} levels.

Rich et al. (23) studied patients with implanted defibrillators in the Boston area, and reported an increased risk of paroxysmal atrial fibrillation episodes associated with short-term increases in ambient ozone. The odds ratio for a 44- $\mu\text{g}/\text{m}^3$ increase in ozone during the hour before the arrhythmia was 2.1 (95% CI 1.2–3.5; $P = 0.001$). The associations with $\text{PM}_{2.5}$, nitrogen dioxide and black carbon were not significant.

These first studies of acute changes in cardiac function associated with exposure to ambient ozone provide biological plausibility for the associations between cardiac morbidity and mortality and ozone levels in the epidemiological studies. Nevertheless, in 13 out of 19 studies focusing on hospital admissions for cardiovascular diseases, no significant effects of ozone were observed. Some of these studies were reviewed in the update of the air quality guidelines (2). The more recent studies from France (24), New Zealand (25) and the United States (Boston) (26) all conclude that ozone was not associated with cardiovascular morbidity. Several of the negative studies did not, however, include an adjustment for the negative correlation between primary pollutants emitted during combustion and ozone, thus limiting their ability to detect a positive association.

Mortality

The results of some representative studies relating ozone to mortality are summarized in the WHO air quality guidelines (Annex 1, Table 3) (2). Significant associations were obtained for different causes, mainly respiratory and (to a lesser extent) cardiovascular. The effects of ozone on mortality were detected mostly in the elderly, and the studies focusing on mortality in children are not fully coherent. Interestingly, in Asia, ozone was associated with mortality due to stroke (27). The magnitude of the mortality risk exhibited a seasonal variation, being more intense in warmer weather. The range of the relative risk of mortality due to respiratory diseases for an increase of 20 $\mu\text{g}/\text{m}^3$ ozone was between 1.0023% (28) and 1.066% (29), such variation depending on age group, season and model specifications. It is reasonable to postulate that adjusting the models for tempera-

ture plays a significant role in the magnitude of the coefficients relating ozone to mortality. The relationship between acute effects of ozone and mortality was reinforced by the recent publication of four meta-analyses (16,30–32). These were consistent in showing a significant association between ozone and short-term mortality that was not substantially altered by exposure to other pollutants (including PM), temperature, weather, season or modelling strategy. Increases in total mortality have been observed at concentrations as low as 50–60 $\mu\text{g}/\text{m}^3$ (1-hour average) (30).

The meta-analysis of European studies published between 1996 and 2001 on short-term effects of ozone on all non-accidental causes of death at all ages (or older than 65 years) resulted in relative risks per 10- $\mu\text{g}/\text{m}^3$ increase in ozone of 1.003 for all-cause, 1.004 for cardiovascular and 1.000 for respiratory mortality (CIs shown in Table 2.1) (21). In each group, the estimates are based on studies in France, Italy, the Netherlands, Spain and the United Kingdom. More recent meta-analyses, based on larger sets of studies, collectively demonstrate short-term associations between ozone and mortality, although the estimates of relative risk vary between cities (16,31,32). The excess risk estimates were higher in summer (when ozone levels are high and people spend more time outdoors) and lower or null in cold seasons (when ozone levels are low and exposures are expected to be low).

In the time-series studies, especially more recent ones, the ozone effect is usually adjusted for both temperature and season. Thus the stronger effects of ozone reported for the summer season may largely be explained by negative correlation between ozone and locally emitted combustion products (as traffic exhaust) in winter. In the APHEA2 study, the effect of ozone in winter was as strong as that in summer, if carbon monoxide was adjusted for (30).

A recent analysis of the effects of ozone on mortality in 48 cities in the United States studied a hypothesis that deaths associated with exposure move the time of death by only a short time (mortality displacement) (34). Analysing the lag structure of mortality in a time-series model, the authors demonstrated that the effect of exposure was larger (0.5% per 10 ppb ozone, 8-hour average) for deaths occurring on days 0–3 after exposure than on the day of exposure alone (0.3%). Further, there was no effect on mortality in the following period. This study demonstrates that risk assessments using a single day of ozone exposure are likely to underestimate, rather than overestimate, the public health impact.

Heatwaves and ozone

During August 2003, high temperatures were observed in western Europe. France was the country most affected, with around 15 000 excess deaths. Questions then arose about the contribution of elevated ozone concentrations to the health impact during the heatwave. In the follow-up period, several studies were conducted to investigate the relationships between temperature, photochemical

air pollution and mortality during the period 1996–2003, including the heatwave. The specific contribution of ozone was assessed.

The French study included nine cities covered by the French surveillance system on air pollution and health (PSAS-9) that were also involved in the European APHEIS programme (35). Ambient ozone concentrations were collected from local air quality monitoring networks. In Paris, for example, the median ozone level (maximum daily 8-hour average) between 1 June and 30 September 2003 was 93 $\mu\text{g}/\text{m}^3$ (25th and 75th percentiles 70 $\mu\text{g}/\text{m}^3$ and 122 $\mu\text{g}/\text{m}^3$, respectively). The highest values were measured in Marseilles (median 123 $\mu\text{g}/\text{m}^3$, 25th and 75th percentiles 104 $\mu\text{g}/\text{m}^3$ and 137 $\mu\text{g}/\text{m}^3$, respectively). Short-term excess risks of total mortality linked to ozone were assessed for the 1996–2003 period (including the heatwave) and compared to this indicator for the 1990–1997 period (without the heatwave). The pooled excess risk increased moderately between the two periods (1.01%, 95% CI 0.58–1.44 vs 0.66%, 95% CI 0.34–0.97 per 10 $\mu\text{g}/\text{m}^3$ of ozone) but local estimates varied significantly between the cities. For the period 3–17 August 2003, the excess risk of deaths linked to ozone and temperatures together ranged from 10.6% in Le Havre to 174.7% in Paris. The relative contributions of ozone in this combined effect varied among the cities, ranging from 2.5% in Bordeaux to 85.3% in Toulouse. The number of attributed deaths per 100 000 inhabitants ranged from 0.9 in Lyon to 5.5 in Toulouse. For the nine cities, the total number of deaths attributable to ozone exposure was 379.

In the Netherlands, an excess of 1000–1400 deaths was attributed to the high temperatures during the 2003 heatwave (31 July–13 August). Fischer et al. (36) estimated the number of deaths attributable to the ozone and PM_{10} concentrations in the Netherlands during the period June–August 2003 and compared the results with estimates for previous summer periods (2000 and 2002). The effects of ozone and of PM_{10} are considered to vary independently in the summer and thus to be additive. An excess of around 400 ozone-related deaths may have occurred during the 2003 heatwave compared to an “average” summer in the Netherlands (Table 2.2).

Table 2.2. Estimated numbers of ozone- and PM_{10} -related deaths in the summers of 2000, 2002 and 2003 in the Netherlands

Period	Estimated deaths related to ozone (95% CI)	Estimated deaths related to PM_{10} (95% CI)
June–August 2000	990 (700–1260)	1290 (640–1930)
June–August 2002	1140 (820–1460)	1380 (690–2060)
June–August 2003	1400 (1000–1780)	1460 (730–2180)
Excess 2003 vs 2000	410 (380–450)	160 (70–260)
Excess 2003 vs 2002	250 (220–290)	80 (–20–180)

Source: Fischer et al. (36).

In the United Kingdom, Stedman (37) calculated excess deaths related to air pollution during the August 2003 heatwave, during which temperatures peaked at a new record of 38.5 °C. The Office for National Statistics reported an excess of 2045 deaths in England and Wales for the period 4–13 August 2003 above the 1998–2002 average for that time of year. Stedman used dose–response functions from times-series epidemiological studies recommended by the Committee on the Medical Effects of Air Pollution (COMEAP). For ozone, the number of deaths was calculated with and without a health effect threshold (100 µg/m³). Stedman estimated that there were between 225 and 593 excess deaths in England and Wales during these first two weeks of August 2003 associated with elevated ambient ozone concentrations. For PM₁₀, 207 excess deaths were estimated to have occurred during this period. This represents (for ozone and PM₁₀) 21–38% of the total excess deaths.

All these studies, despite geographical differences, tend to show that a non-negligible proportion of the excess deaths during heatwaves is associated with elevated concentrations of air pollutants, including ozone, independently from the direct effect of high temperatures.

Chronic effects in humans

Ideally, an assessment of long-term effects of ozone in humans would include epidemiological studies investigating cumulative ozone exposure in association with three interrelated types of outcome, namely associations with: (a) early markers of chronic processes relevant to the development of disease; (b) onset or incidence of chronic diseases; and (c) reductions in life expectancy.

Lung function of children and young adults

Measures of lung function have most often been used as an objective early marker of chronic pulmonary effects. Given the lifetime pattern of growth and decline in lung function, both cross-sectional and prospective studies can provide insight into the role of ozone exposure. The former approach has been used in children, adolescents and young adults. Prospective studies have been conducted in children and adolescents, focusing on lung function growth. Decline in lung function has not yet been investigated in relation to cumulative exposure to ozone.

The most thorough study is the Children's Health Study, carried out in multiple cohorts in 12 communities in southern California (38). The cross-sectional analyses indicated associations between lung function and annual means of daily 1-hour ozone maxima. An association with small airway function was particularly pronounced (39). However, the findings were significant only among girls and in boys spending more time outdoors. For the same cohorts studied prospectively, lung function growth rates showed significant associations with a set of urban pollutants (PM_{2.5}, nitrogen dioxide and acid vapour) but findings for ozone

were not significant and were inconsistent across age groups and lung function parameters (40–42). Growth rates in small airway function – primarily expected to be associated with ozone – were inversely associated with ozone among the youngest cohort only (41) but not in the eight-year follow-up from age 10 to 18 years (42).

The null findings in the growth rate analyses do not necessarily contradict positive findings in the cross-sectional analysis. The study was limited to 12 communities with only a two-fold range in ambient ozone levels and within-community variation in personal exposure owing to differences in the use of air conditioning. Ventilation patterns and time spent indoors were not fully controlled for and may be a source of noise or bias. Further, if the chronic effects of ozone happened primarily in early life, one may expect discrepancies between cross-sectional results and those based on growth rates if the latter were observed after the susceptible period.

Two studies carried out by the University of California at Berkeley used a powerful cross-sectional design to maximize lifetime exposure to ozone. Instead of selecting (a limited number of) communities, freshmen who had lived all their lives in California were invited to participate. The pilot study (43) included 130 and the main study (44) 255 nonasthmatic students. Ozone was interpolated on a monthly basis to each residential location over their lifetimes. The integration of time–activity data into the exposure model did not affect the results. Both studies observed consistent and significant cross-sectional associations between individual lifetime ozone exposure and, in particular, small airway function, namely FEF_{25-75} and FEF_{75} (but also FEV_1) at age 18–20 years. A contrast of $2 \mu\text{g}/\text{m}^3$ in lifetime 8-hour average ozone was associated with 2.7% and 2.9% lower FEF_{75} in males and females, respectively (44). The main study was large enough to investigate susceptible subgroups, and revealed that significant effects occurred only among students with small airways (marked by the ratio FEF_{25-75}/FVC) (44). Effects were robust to adjustment for co-pollutants (PM and nitrogen dioxide).

Galizia & Kinney (45) employed a similar design, with individual assignment of long-term exposure to Yale (New Haven) College freshmen who had geographically diverse residential histories. FEV_1 and FEF_{25-75} were significantly (and FEF_{75} borderline) associated with ozone exposure. FEF_{25-75} was 8.11% (range 2.32–13.9%) different between the lowest and highest exposure levels ($\sim 300 \mu\text{g}/\text{m}^3$, long-term average of daily 1-hour maximum). Stratified analyses showed effects to be stronger in men but not significant in women. Another study addressing seasonal exposure was that of Ihorst et al. (46), who made lung function measurements twice a year over $3\frac{1}{2}$ years on 2153 schoolchildren in 15 towns in Austria and Germany. They concluded that ozone exposure may be related to seasonal changes in lung function growth, but are not detectable over $3\frac{1}{2}$ years owing to partial reversibility or to the relatively low concentrations of ozone.

Atherosclerosis and onset of asthma

A novel marker of chronic preclinical damage has been used to investigate effects of air pollution on atherosclerosis, measured by carotid intima-media thickness (CIMT) (47). Systemic inflammatory responses to oxidant pollutants may contribute to atherogenesis. A Los Angeles study reported cross-sectional associations between CIMT and residential outdoor PM_{2.5} levels, whereas associations with residential outdoor ozone were weak and statistically not significant (48).

The onset of asthma (new diagnosis) was prospectively investigated in the Children's Health Study (49) and in adults in the Adventist Health and Smog (AHSMOG) Study (50). The Children's Health Study followed more than 3500 nonasthmatic children aged 9–16 years from 1993 to 1998. Community mean ozone level was not associated with new a diagnosis of asthma. However, the number of outdoor sports engaged in by the children was correlated with asthma onset in communities with a high level of ozone. Playing three or more outdoor sports was associated with a relative risk of 3.3 (range 1.9–5.8) for developing asthma. In contrast, physical activity was not a risk for asthma in low-ozone communities (49).

The 15-year follow-up of the AHSMOG cohort included 3091 non-smoking adults (50). As in the University of California studies, ozone levels were interpolated to residential locations to assign a 20-year exposure history to each subject. A 54- $\mu\text{g}/\text{m}^3$ change in long-term ozone was associated with a two-fold risk for asthma onset among men, though not among women. One may speculate that women spent more time indoors (where ozone levels are very low) or that protective hormonal factors may play a role. The interaction may also be a chance finding.

Cross-sectional retrospective assessments of symptom prevalence (e.g. wheezing) may not necessarily reflect long-term effects but rather the accumulated period prevalence of cumulative acute effects (such as acute exacerbations of asthma). Thus retrospective studies are not reviewed here, as they cannot distinguish acute from chronic effects.

Reduction in life expectancy

Cohort mortality studies cannot unambiguously distinguish between (a) effects that lead to chronic processes and diseases that shorten life (i.e. chronic effects) and (b) acute or subacute effects of exposure that lead to death (51). Cohort studies capture, at least in theory, both effects. Thus the effects observed in cohort studies may not necessarily be solely due to chronic exposures.

Several cohort studies have reported associations between long-term mean concentrations of ambient air pollutants and death rates, but results for ozone were not consistent, not as rigorously investigated as those for PM, or not reported at all. The American Cancer Society (ACS) study – the largest cohort of all – and the Harvard Six City study found no significant association of ozone

with mortality (52,53). The reanalysis by the Health Effects Institute reported, however, a significant association of “warm-weather ozone” and cardiopulmonary mortality, with a relative risk of 1.08 (range 1.01–1.16) (54) and a significantly “protective” association for lung cancer. The recent extended analysis of the ACS cohort (55) observed increased mortality from cardiopulmonary diseases (though not statistically significantly) associated with long-term summertime exposure to ozone. No increase was seen for risk of death from lung cancer. A more recent analysis conducted among ACS participants from southern California, however, observed no effect of long-term exposure on either cardiopulmonary disease or lung cancer mortality (56). The two studies differ in that the southern California study based its exposure assignment on geospatially-derived estimates of residential concentrations, whereas the national study assessed exposure at an urban area level.

In the 15-year follow-up of the AHSMOG population, lung cancer was significantly associated with ozone level among men (57). Associations were positive for other causes but not statistically significant.

Uncertainties in long-term effects of ozone

This question needs to be addressed in all the studies cited above. In contrast to pollutants such as PM, ozone is highly reactive. As a consequence, indoor : outdoor ratios are in general low and very heterogeneous across houses, locations and seasons. This spatio-microenvironmental heterogeneity is far more critical for ozone than for PM. Some studies conducted on the east coast of the United States suggest that ambient ozone concentrations may be very poorly associated with personal exposure (or dose), at least in some cities and/or seasons (58,59). This has not been investigated in any of the locations of the chronic effect studies cited above.

Pollutants such as PM and related primary pollutants (e.g. nitrogen oxides) react with ozone, leading to (usually unmeasured) negative correlations between (personal) exposure to ozone and other pollutants. These other pollutants may also contribute to adverse health effects; the ability to observe the long-term effects of ozone may thus be a major methodological challenge, particularly if the exposure term used to characterize ozone exposure was less correlated with personal ozone than might be the case for these other pollutants. Community-based single-monitor studies (i.e. with clustered study populations) are more affected by these sources of error and noise than subject-based designs with individual assignment of exposure, such as the University of California and AHSMOG studies.

The interaction of outdoor activity, ozone level and asthma observed in the Children’s Health Study (and possibly in men in the AHSMOG study) also indicates that time spent outdoors needs to be controlled in the exposure assessment. Given prevailing lifestyles, with over 90% of time spent indoors with generally

low concentrations, time spent outdoors (and in outdoor activity) becomes the most important determinant of exposure to high ozone levels.

The issue of thresholds of no effect has yet to be addressed in studies of chronic effects. Ozone is a natural constituent of the atmosphere and the lung is equipped with oxidant defence mechanisms, and one may speculate that some levels of no effect may exist. An early cross-sectional investigation with NHANES II data observed inverse associations of ozone, nitrogen dioxide and total suspended particulates with FVC and FEV₁ among people 6–24 years of age (60). The pattern in these associations with ozone would support speculation about thresholds of no effect. The results were driven by data from Californian communities in the upper range of the ozone distribution.

Conclusions of chronic effect studies

Evidence for the chronic effects of ozone has become stronger. Animal data and some autopsy studies indicate that chronic exposure to ozone induces significant changes in airways at the level of the terminal and respiratory bronchioli. Epidemiological evidence of chronic effects is less conclusive, owing mostly to an absence of studies designed specifically to address this question and inherent limitations in characterizing exposure. The studies with the most efficient approaches and more individual assignment of exposure provide new evidence for chronic effects of ozone on small airway function and possibly on asthma. Substantial uncertainties remain, however, and need to be addressed in future investigations. The partly inconsistent patterns or lack of associations may originate from limitations in exposure assessment and/or from an inability to identify those most susceptible to the chronic effects of ozone. They should thus not be interpreted as evidence of no adverse chronic effects following repeated daily and seasonal exposure to ozone

Thresholds

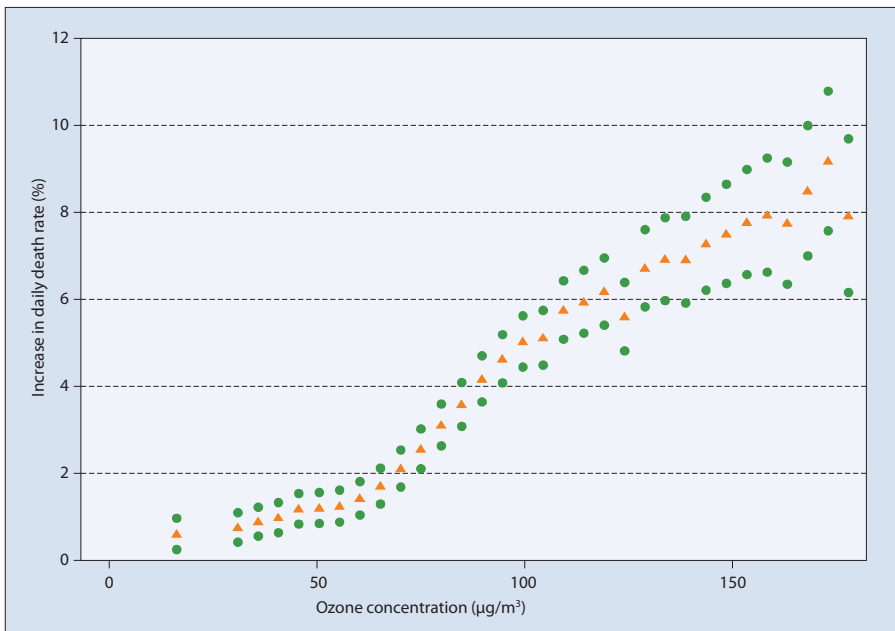
Since the human respiratory tract contains antioxidant defences, and it has been shown that such defences are consumed during ozone exposure, it is reasonable to propose the existence of a threshold in the dose–response functions relating ozone to adverse health effects. In other words, effects should occur mainly after the depletion of antioxidant defences. The concept of a threshold is supported by studies dealing with controlled exposures. In epidemiological studies, however, the evidence of a threshold is weaker, owing probably to the fact that variable individual thresholds become less evident at the population level. In other words, it is highly likely that it will be impossible to ensure a concentration of no effect in a population. The diversity of factors possibly determining the individual threshold, such as age, pre-existing diseases, social and economic status, habits and genetics, will obscure the determination of a clear no-effect concentration.

The European APHEA2 study, based on data from 23 cities, examined the shape of the association between ozone levels and risk of dying (30). The effects were found mostly in the summer, when the relationship between ozone and mortality does not seem to deviate significantly from linearity, and a significant increase in risk was estimated for ozone concentrations above a 1-hour average of 50–60 $\mu\text{g}/\text{m}^3$ (Fig. 2.2).

A United States study recently investigated possible alternative dose–response functions using ozone and mortality data from 98 cities (61). The investigators found that any safe threshold, if one exists, would be far below the levels set out in current ozone standards and guidelines. The central estimate for same-day and previous-day averages, for example, deviated from the no-effect line above the 40- $\mu\text{g}/\text{m}^3$ level (Fig. 2.3). The risk estimates were statistically significant above 80 $\mu\text{g}/\text{m}^3$ and were stable for concentrations over 70 $\mu\text{g}/\text{m}^3$. The analysis used “daily mean ozone level” as the indicator of exposure. When a ratio of 15 : 8 between the 8-hour and daily means is applied to adjust between various averaging times, the above results indicate no effects at 75 $\mu\text{g}/\text{m}^3$ (8-hour average) and statistically significant effects at 150 $\mu\text{g}/\text{m}^3$ (8-hour average).

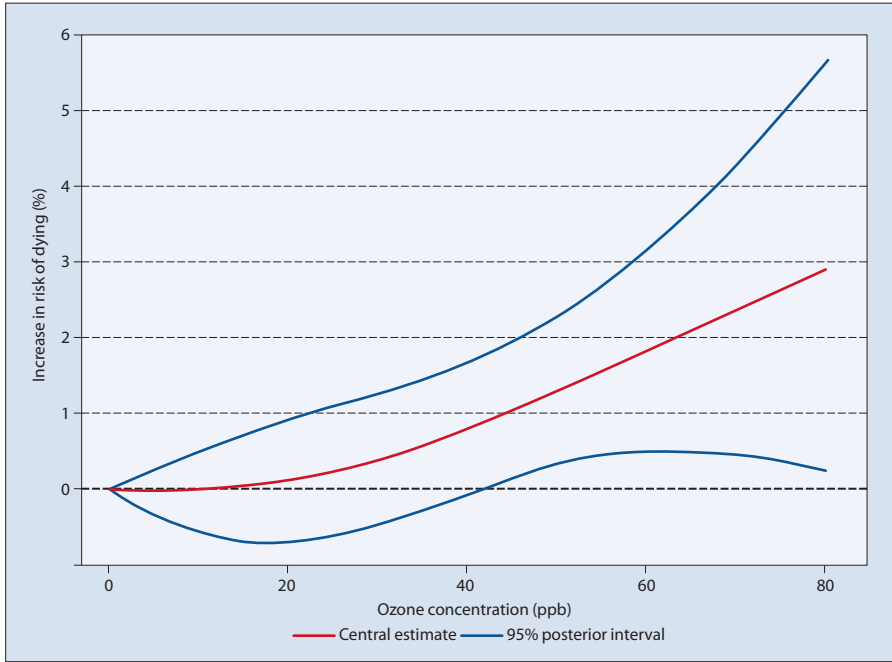
Overall, recent epidemiological studies provide consistent evidence that daily changes in ambient ozone exposure are linked to premature mortality even at

Fig. 2.2. The relationship between ozone concentration (maximum daily 1-hour average) and the daily death rate (average of lags 0 and 1) during the summer, based on data from 23 European cities in the APHEA2 study



Note: The green dots show 95% CI limits.
Source: Gryparis et al. (30).

Fig. 2.3. The relationship between ozone concentration (daily average) and the daily death rate (average of lags 0 and 1), based on data from 98 urban communities in the United States



Source: Bell et al. (61).

very low pollution levels, without clear evidence for a threshold of effects within the range of exposures observed in urban communities in both Europe and North America. The confidence related to the magnitude of the risk increases above 50–70 $\mu\text{g}/\text{m}^3$.

Susceptible groups

Individuals vary in their ozone responsiveness for different outcomes. Airways symptoms, wheeze, chest tightness, cough and asthma are associated with ozone exposure and individuals with underlying lung or airways diseases are therefore at higher risk of being affected by ozone exposures. The overall health status of an individual plays a role in the sensitivity; dietary conditions such as general malnutrition or deficiency of specific nutrients or vitamins may increase the sensitivity of individuals.

Recent studies have shown that abnormalities of the members of the glutathione S-transferase superfamily (*GSTM1*, *GSTT1* and *GSTP1*) can affect responses of children to oxidant air pollutants. It appears that the effects of ozone exposure on symptoms are greater in asthmatic children. Lung function decrements are more consistent in asthmatic children, especially those with low birth weight. Children may also be exposed to a greater extent than adults because of their

greater physical activity and likelihood that they spend a larger part of the day outdoors. The higher metabolic rate of children, revealed in a higher breathing volume per unit mass, also increases the internal dose of pollutants for a given ambient concentration. This point applies also to athletes and others exercising outdoors.

Besides increasing the ozone dose, a higher ventilation rate increases the penetration of ozone deep into the lungs, since the tidal volume also increases. Duration of exposure is also a critical factor: the effects of ozone accumulate over many hours, but after several days of repeated exposure there is adaptation in functional though not inflammatory responses. The effects of ozone exposure on lung function, symptoms and school absences are larger in children who exercise more or spend more time outdoors.

There is some evidence that short-term effects of ozone on mortality and hospital admissions increase with age. Gender differences are not consistent.

Health implications

The adverse effects of ozone on the respiratory tract, from the nasal passages to the gas-exchange areas, are unequivocal. Although there are considerable variations in response between species and between individuals, acute ozone exposure causes reduced pulmonary function, pulmonary inflammation, increased airway

permeability and heightened hyperactivity. These effects and ensuing tissue injury in the small airways and the gas exchange region, depending on exposure concentration and duration as well as individual susceptibility, may lead to irreversible changes in the airways and worsen lung disease. The evidence for cardiovascular effects is less conclusive.

Evidence for the chronic effects of ozone is supported by human and experimental information. Animal data and some autopsy studies indicate that chronic exposure to ozone induces significant changes in airways at the level of the terminal and respiratory bronchioli. The reversibility (or not) of such lesions is a point that deserves clarification. Epidemiological evidence of chronic effects is less conclusive, owing mostly to an absence

Box 2.1. WHO air quality guidelines and European Union (EU) ozone standards for protection of health

WHO air quality guideline for ozone (2)

- Guideline: maximum daily 8-hour average $100 \mu\text{g}/\text{m}^3$
- Interim target (IT-1): maximum daily 8-hour average $160 \mu\text{g}/\text{m}^3$
- High level: maximum daily 8-hour average $240 \mu\text{g}/\text{m}^3$

EU air quality directive (62)

- Target value: maximum daily 8-hour average $120 \mu\text{g}/\text{m}^3$, not to be exceeded on more than 25 days per calendar year (to be met by 1 January 2010)
- Long-term objective: maximum daily 8-hour average within a calendar year $120 \mu\text{g}/\text{m}^3$
- Information threshold (1-hour average): $180 \mu\text{g}/\text{m}^3$
- Alert threshold (1-hour average): $240 \mu\text{g}/\text{m}^3$

of studies designed specifically to address this question and inherent limitations in characterizing exposure. The studies with the most efficient approaches and more individual assignment of exposure provide new evidence for chronic effects of ozone on small airway function and possibly on asthma.

Based on the accumulated evidence, WHO has recently updated the air quality guideline for ozone, setting it at 100 $\mu\text{g}/\text{m}^3$ for a daily maximum 8-hour average (Box 2.1). It is possible that health effects will occur below this level in some sensitive individuals. The discussion on the update of the air quality guidelines concluded that, based on time-series studies, the number of attributable deaths brought forward can be estimated at 1–2% on days when the ozone concentration reaches this guideline level. The results of the European APHEA2 study suggest that this increase might be even greater.

3. Sources of ozone precursors

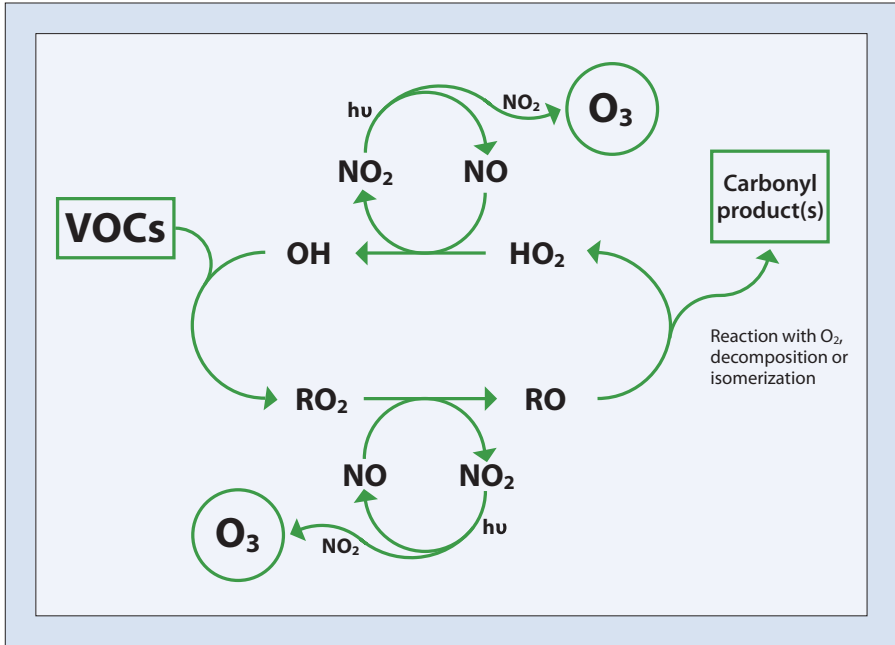
KEY MESSAGES

- Ozone is a secondary pollutant formed in photochemical reactions from nitrogen oxides (NO_x) and volatile organic compounds (VOCs) as well as methane and carbon monoxide. The process of ozone formation is complex and depends on sunlight, geographical factors and the availability of the precursors.
- In the vicinity of the source, NO_x deplete ozone, leading to lower levels of ozone in urban areas. Downwind, at a distance from the source, however, NO_x emissions lead to ozone formation. Ozone has a strong hemispheric component, typically reaching 20–40 ppb (40–80 µg/m³).
- The majority of ozone precursor emissions originate from anthropogenic sources. Important human activities that contribute to ozone formation include transport (especially road vehicles and international maritime shipping), combustion processes in energy production and industry, solvent use, biomass burning and agricultural practices.
- Owing to the presence of stringent emission control legislation, ozone precursor emissions are expected to decline in the EU over the coming decade. However, lack of equivalent legislation will not prevent further increases in precursor emissions in other countries that are Parties to the Convention on LRTAP. This growth in emissions is expected to increase hemispheric ozone background concentrations. Furthermore, climate change could lead to higher biogenic emissions in the future.
- Methane emissions promote ozone formation and global climate change.
- Measures to reduce ozone precursor emissions will have many health benefits in addition to the direct health impacts of lower ozone levels. These measures will also reduce levels of other hazardous air pollutants and greenhouse gases and will reduce radiative forcing. At the same time, less ozone in the atmosphere will result in less damage to vegetation.

Ozone formation and atmospheric transport

Ozone is the most important oxidant in the troposphere. It is formed by photochemical reactions in the presence of sunlight and precursor pollutants such as NO_x, VOCs, methane and carbon monoxide (Fig. 3.1). The lowest annual average tropospheric ozone concentrations in remote background areas in Europe have ranged between 40 and 90 µg/m³ (63). Ozone levels experienced at a certain location are influenced by (a) the hemispheric concentrations of ozone in the free troposphere resulting from emissions from the northern hemisphere; (b) the ozone generated by long-range transport of the precursor over some several hun-

Fig. 3.1. Schematic representation of the photochemical formation of ozone in the presence of VOCs and NO_x



Source: Jenkin & Hayman (64).

dred to thousands of kilometres; (c) locally increased ozone production downwind of sources of precursor emissions in sunny weather; (d) local destruction of ozone (titration) due to nearby NO_x emissions (particularly important at sites close to high NO_x emissions, i.e. in urban areas); and (e) deposition of ozone to the ground.

The lifetimes of many ozone precursors and their conversion products are sufficiently long to allow them to be transported over long distances in the atmosphere. Consequently, the large-scale ozone “background” level has a strong long-range transport component determined by a wide range of emission sources (63). On the other hand, the aforementioned factors (plumes, titration and deposition) depend strongly on small-scale geographical and meteorological conditions and superimpose local variations on the large-scale background level. Local emissions in urban areas reduce ozone levels close to the source and increase levels in the downwind plume. Local variation in deposition rates is also an important factor affecting the lifetime and local concentration of ozone.

As a result of anthropogenic emissions and photochemical reactions, ozone displays strong seasonal and diurnal patterns in urban areas, with higher concentrations in summer and in the afternoon. The correlation of ozone with other pollutants varies by season and location. These unique features of the atmospheric chemistry of ozone make the interpretation of the shape of exposure–response

relationships particularly complex. The formation of ozone is temperature-dependent, so that the high end of the exposure–response relationship will be based on hot sunny summer days and the lower end on winter days. Unfortunately, this may mean that factors other than the ozone concentration are varying across the range of the exposure–response relationship. In the eastern United States, for example, ozone is often positively correlated with particles in the summer and negatively correlated with particles in the winter (65). Ozone can be particularly low in cold inversion conditions when other pollutants accumulate.

Sources of ozone precursor emissions

Ozone precursor gases are emitted from a wide variety of anthropogenic and natural sources.

At present, the most important source of anthropogenic NO_x emissions on the global scale is road transport (29% in 2000), followed by combustion in power plants and industry (27%). Some 17% of global emissions come from international maritime shipping, 10% from non-road vehicles and 2% from aircraft. Open burning of biomass due to forest fires, savannah burning and agricultural practices accounts for approximately 15% of global anthropogenic emissions. Natural sources include soils and lightning.

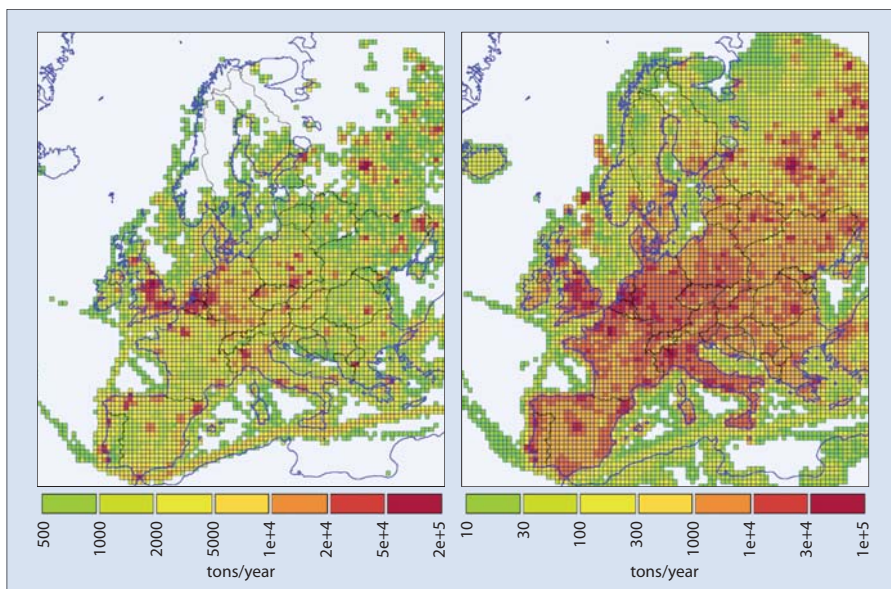
There are a large number of **non-methane VOCs** in the atmosphere that contribute to ozone formation. Important anthropogenic sources include incomplete combustion of fossil fuels, evaporative losses of fuels, solvent use, various industrial production processes, agricultural activities and biomass burning. Globally, however, it is believed that natural sources of VOCs far outweigh anthropogenic sources.

On a global scale, emissions of **carbon monoxide** from deforestation, savannah burning and the burning of agricultural waste account for about half of anthropogenic emissions. The rest come from fuel combustion, with a quarter from household solid fuels and about 20% from road transport. The primary natural sources of carbon monoxide are vegetation, oceans and wildfires (biomass burning).

Globally, most **methane** emissions are anthropogenic, with an important fraction of biogenic emissions directly connected to human activities such as rice cultivation. The major anthropogenic sources include coal mining, the gas and oil industries, landfill, ruminant animals, rice cultivation and biomass burning. The single largest natural source of methane is wetlands.

The environmental impacts of emissions are critically influenced by the availability of the various pollutants in ambient air. In addition to other factors, spatial emission densities are important determinants of pollutant concentrations in ambient air. As shown in Fig. 3.2, there are substantial differences in emission densities of NO_x and non-methane VOCs across Europe, inter alia as a consequence of different population densities.

Fig. 3.2. Density of NO_x emissions (left) and non-methane VOC emissions (right) in Europe in 2004



Source: Isaksson et al. (66).

Projections of future emissions of ozone precursors

Ozone precursor emissions are expected to change significantly as a result of population growth, economic development, technological progress and uptake, control measures, varying land use, and climate and other environmental changes. Scenarios are often used to analyse how different drivers may affect future emission rates and to assess the associated uncertainties. These are usually based on a mixture of quantitative information and expert judgement, and aim at producing an internally coherent picture of how the future could develop for a given set of explicit assumptions.

Modelling tools are frequently used to develop coherent pictures of how developments of the different factors will influence future emissions. This chapter summarizes baseline projections of ozone precursor emissions that have been developed within the CAFE programme (67) with the Regional Air Pollution Information and Simulation (RAINS) model.

The RAINS model, developed by the International Institute for Applied Systems Analysis (IIASA), combines information on economic and energy development, emission control potentials and costs, atmospheric dispersion characteristics and environmental sensitivities towards air pollution (68). The model is able to address threats to human health posed by fine particulates and ground-level ozone, as well as the risk of damage to ecosystems from acidification, excess nitrogen deposition (eutrophication) and exposure to elevated ambient levels of ozone. These problems related to ozone air pollution are considered in a multi-

pollutant context, quantifying the contributions of NO_x and non-methane VOCs. Over the last few years, the RAINS model has been extended to address synergies between air pollution control and greenhouse gas mitigation. The new GAINS (Greenhouse gas – Air pollution INteractions and Synergies) model addresses, in addition to the five air pollutants, emissions of the six greenhouse gases included in the Kyoto Protocol.

The CAFE programme aimed at a comprehensive assessment of the available measures for further improving European air quality beyond the achievements expected from the full implementation of all existing air quality legislation. The EU has established a comprehensive legislative framework that allows for economic development while moving towards sustainable air quality. EU directives specify minimum requirements for emission controls from specific sources. The CAFE baseline assessment quantifies for each Member State the impacts of the legislation on future emissions.

The CAFE baseline projections assumed current trends in economic development, taking into account the effects of tightened emission control legislation. Two major scenarios were analysed. The first baseline projection, based on current legislation (the CLE scenario), adopted as a starting point the baseline energy projection of the Directorate General for Energy and Transport of the European Commission (69) and did not assume any further climate measures beyond those already adopted in 2002. Alternatively, a second, “maximum technically feasible reductions” (MTFR) scenario attempted to quantify how the decarbonization of the energy system would take place owing to climate policies based on guidance received from the Directorate General for the Environment, without prejudging the actual implementation of the Kyoto Protocol and of possible post-Kyoto regimes (70).

For the MTFR scenario, NO_x emissions from the EU15¹ are expected to decline by 31% in 2010 and by 48% in 2020 compared to 2000 (Fig. 3.3). The largest reductions will result from measures in the power generation sector (–44% in 2010) and for mobile sources (–35% in 2010). For the 10 new Member States that joined in 2004, NO_x emissions are computed to decline by 33% in 2010 and by 57% in 2020. The CLE scenario yields slightly lower reductions, with –46% for EU15 and –54% for the new Member States in 2020.

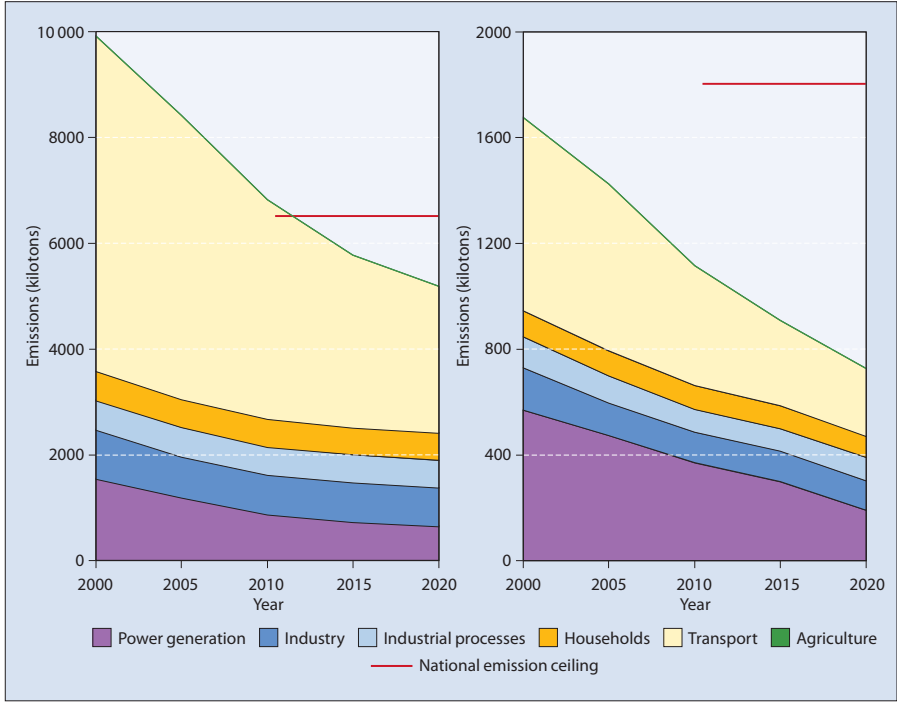
The CAFE 2020 baseline projections for NO_x indicate a significant shift in the contributions made by the individual source categories to total NO_x emissions in EU25² (Fig. 3.4 and 3.5). Owing to strict emission controls for vehicles, the share of NO_x emissions caused by mobile sources will decline from 60% in 2000 to less than 50% in 2020. Especially efficient are the controls on petrol engines, so that their contribution to total NO_x emissions will shrink from 17% in 2000 to only

¹ The European Union with 15 Member States as it existed between 1995 and 2004.

² The European Union with 25 Member States as it existed between 2004 and 2007.

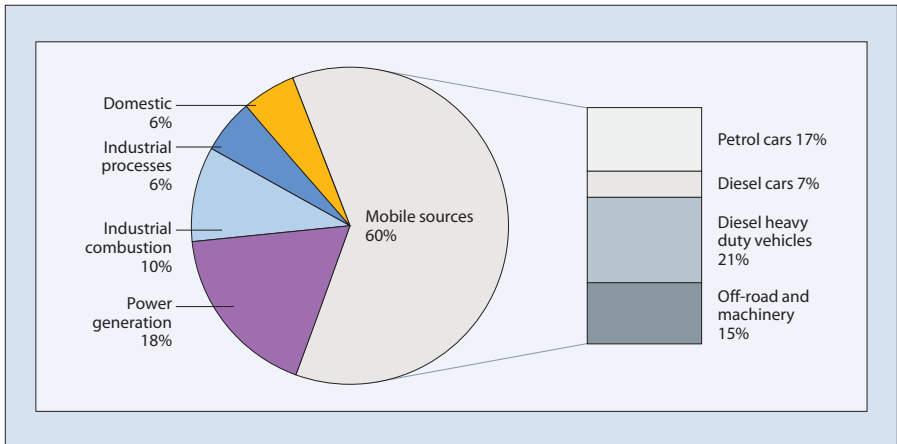
4% in 2020. For 2020, 18% of NO_x emissions are calculated to emerge from heavy duty diesel engines, while the share from off-road mobile sources will increase to 19%.

Fig. 3.3. NO_x emissions by sector for the EU15 (left) and the 10 new Member States that joined the EU in 2004 (right) for the MTR scenario



Source: Amann et al. (70).

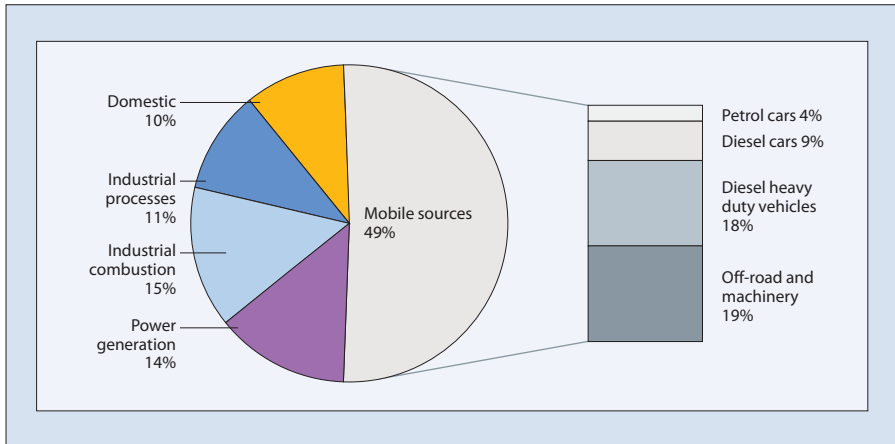
Fig. 3.4. Contributions to NO_x emissions in EU25 in 2000



Source: Amann et al. (70).

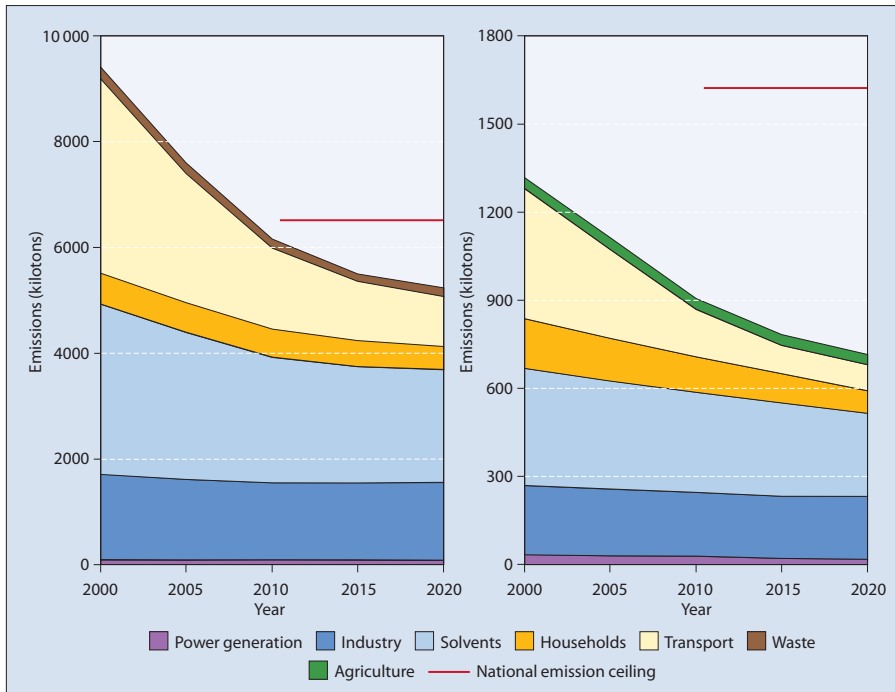
Under the CLE scenario and with the emission control legislation, VOC emissions are expected to decrease in EU15 by 33% in 2010 and 41% in 2020 compared to 2000. There are only minor additional impacts of the MTRF scenario,

Fig. 3.5. Contributions to NO_x emissions in EU25 in 2020 according to the MTRF scenario



Source: Amann et al. (70).

Fig. 3.6. VOC emissions by sector for EU15 (left) and the 10 new Member States that joined the EU in 2004 (right) for the MTRF scenario

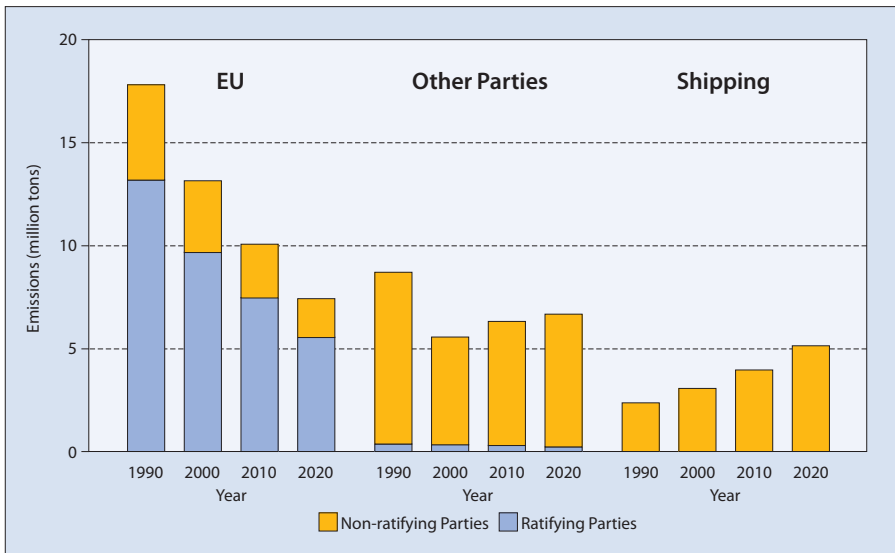


Source: Amann et al. (70).

mainly due to small variations in the transport volumes between the scenarios. In the new Member States, VOC emissions are computed to be 15% lower in 2010 and 33% lower in 2020 than in 2000. In both groups of countries, the decline in emissions from mobile sources supplies the largest contribution to this reduction (Fig. 3.6). While this provisional analysis indicates for some EU15 Member States a potential need for further measures to achieve the emission ceiling, emissions from the whole EU15 would be 3% below the ceiling. However, the new Member States would be 45% under the ceiling.

While ozone precursor emissions from the EU Member States are generally expected to decline in the coming decades, emissions are likely to increase from other countries where less stringent emission controls are in place. Up to 2020, NO_x and VOC emissions from the Parties to the Convention on LRTAP that do not belong to the EU are likely to increase by 15–20% compared to 2000 (Fig. 3.7) (71). Even larger increases have to be anticipated for emissions from international maritime shipping (72).

Fig. 3.7. Projections of NO_x emissions for the Parties to the Convention on LRTAP and shipping

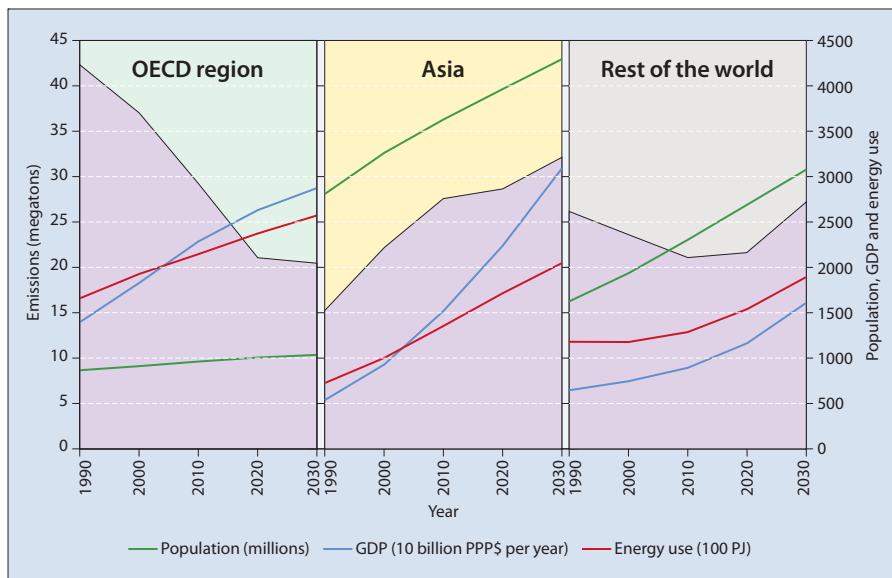


Source: Cofala et al. (71).

As pointed out by, inter alia, the UNECE Task Force on the Hemispheric Transport of Air Pollutants, ozone background levels include a strong intercontinental component that originates from emissions from the entire northern hemisphere (73). Global assessments indicate a continued increase of ozone precursor emissions in developing countries, where even a full implementation of the recently adopted emission control legislation will not be sufficient to compensate for the envisaged increase in economic activities resulting from economic

development (74). While a clear decline in NO_x emissions from the Organisation for Economic Co-operation and Development (OECD) region can be expected, emissions in other parts of the world are likely to increase further (Fig. 3.8).

Fig. 3.8. Global CLE projections of NO_x emissions for the OECD region, Asia and the rest of the world



Source: Cofala et al. (74).

In contrast to NO_x and VOC emissions, for which some reduction measures are currently in place even in developing countries because of concerns about local air quality, methane emissions are expected to grow continuously on a global scale in the absence of more dedicated mitigation policies. Global greenhouse gas scenarios anticipate global anthropogenic methane emissions to grow by between 30% and 60% by 2030 (75).

Global emissions of carbon monoxide from anthropogenic activities are expected to decline in the coming decades owing to the phasing out of domestic solid fuels and the effectiveness of exhaust control measures for vehicles.

While it is difficult to develop robust projections of future emissions of ozone precursors from biogenic sources, the literature points towards a number of mechanisms by which climate change could lead to potentially significant changes in emissions from these sources.

In summary, anthropogenic ozone precursor emissions in industrialized countries are likely to decline in the coming decades, which could have significant impacts on locally generated ozone contributing to peak concentrations. Nevertheless, substantial increases are expected for NO_x and VOC emissions from other

parts of the world with rapidly developing economies, as well as for methane emissions. These could cause a further increase in the hemispheric background concentration of ozone.

Interactions of ozone precursor emissions with other environmental problems

Ozone precursor emissions not only pose threats to human health but also contribute to a range of other environmental problems (Table 3.1). NO_x and VOC emissions are potent precursors of secondary aerosols that are important components of ambient PM. In addition, NO_x emissions contribute to acid deposition, leading to the acidification of terrestrial and aquatic ecosystems, and to excess nitrogen deposition that threatens biodiversity. Direct exposure to elevated concentrations of NO_x , carbon monoxide and VOCs is considered to have harmful effects on health. Methane is a potent greenhouse gas, its radiative forcing ranking second after carbon dioxide.

Health effects are not the only negative effects of ground-level ozone. It also causes damage to vegetation in forests and natural ecosystems, to agricultural crops and to materials. Further, ozone acts as a greenhouse gas with the third largest radiative forcing.

Table 3.1. Interactions between different atmospheric pollutants and environmental problems (emissions of ozone precursors are highlighted)

	PM	Sulfur dioxide	NO_x	VOCs	Ammonia	Carbon monoxide	Carbon dioxide	Methane	Nitrous oxide	F gases
Health effects										
PM	•	•	•	•	•					
ozone			•	•		•		•		
direct effects		•	•	•		•				
Vegetation damage										
ozone			•	•		•		•		
acidification		•	•		•					
eutrophication			•		•					
Radiative forcing										
direct										
via aerosols	•	•	•	•	•		•	•	•	•
via ozone			•	•		•		•		

4. Ozone levels

KEY MESSAGES

- Background ozone levels in the northern hemisphere are close to levels known to be harmful to health.
- Higher levels are observed in the sunny Mediterranean region than in northern Europe.
- Episodic peak ozone levels in northwestern Europe have declined since the 1990s in response to regional pollution controls applied to NO_x and VOCs. However, most recent data do not show a further decrease.
- Exhaust gas catalysts have reduced the extent of ozone scavenging by the NO_x emissions from petrol-engine vehicles. As a result, ozone levels in towns and cities have begun to rise towards those found in the surrounding countryside.
- Long-term average ozone levels have fallen in some areas and increased in the others, owing to the combined effects of regional pollution controls and the increasing hemispheric background.
- Penetration of rural ozone into urban areas is facilitated by the fall in urban air pollution and reduced ozone depletion in densely populated areas in Europe.
- Climate change may lead to increased ozone concentrations.

Monitoring results

More than 1800 operational ozone monitoring stations routinely report to the European Commission and the European Environment Agency (EEA) (76). The high number of stations is primarily a result of the requirements of EU air quality directive 2002/3/EC relating to ozone in ambient air. This directive requires EU Member States to assess the exposure of the population and of crops and natural vegetation (77). As a result, a large number of stations are located in urban or suburban areas where both population density and ozone concentrations are relatively high (in 2003, this represented 42% of stations). In addition, 23% of the stations are characterized as rural or rural background stations, measuring ozone levels representative of a more regional scale. The remainder are characterized as “traffic stations” (19%) or “industrial stations” (10%) or have an unknown classification (7%). The directive requires the ultraviolet photometric method to be used as the reference method, and this method is used at almost all stations. European ozone data are available from the air quality archive AirBase, developed and maintained by EEA (<http://airbase.eionet.eu.int>). Also, a “real time” map of ozone levels is available on the EEA web site (<http://www.eea.europa.eu/maps/ozone/map>).

A second important ozone monitoring network has been formed in the framework of the Convention on LRTAP and coordinated by EMEP (78); all data are

available from the EMEP web site (<http://www.emep.int>). This network focuses on ozone measured at rural background locations. There is a large overlap between the stations reporting to EMEP and to AirBase; the EMEP network, however, includes a number of remote (e.g. mountain) stations that are important for evaluating ozone trends in the free troposphere.

Ozone as a long-range transported secondary pollutant in Europe

Ozone is present at ground level, throughout Europe, on most days of the year. As a general rule, ozone levels are higher in the rural areas surrounding centres of population than in urban background or roadside and kerbside locations. Levels are generally higher in spring and summer than they are in autumn and winter. Peak summertime levels are higher in southern Europe and lower towards the west and north. Levels are generally highest during the afternoon and lowest during the early morning. Ozone concentrations are expressed either in micrograms per cubic metre ($\mu\text{g}/\text{m}^3$) or in parts per billion (ppb), with a conversion factor of $1 \text{ ppb} = 2 \mu\text{g}/\text{m}^3$ at 20°C and 101.3 kPa (see Box 4.1).

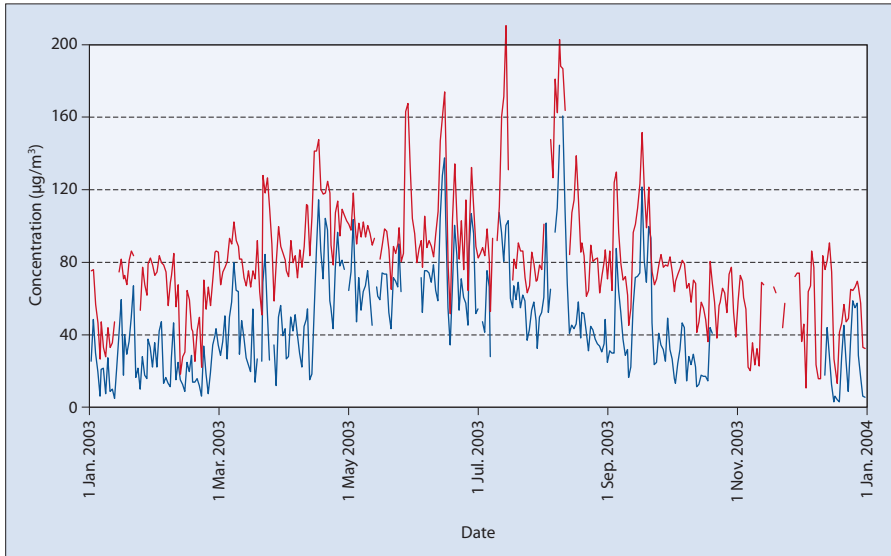
Box 4.1. Mass and volumetric concentrations

The volumetric concentration unit 1 ppb corresponds to 1 mm^3 in 1 m^3 of air. The density of gases varies according to the molecular weight, but also according to the pressure and temperature. Therefore the ratio of volumetric to mass concentration varies accordingly. At normal atmospheric pressure, 1 ppb ozone corresponds to a mass concentration of $2.0 \mu\text{g}/\text{m}^3$ at 20°C or $2.1 \mu\text{g}/\text{m}^3$ at 0°C .

Fig. 4.1 presents the maximum daily 8-hour average ozone concentrations for each day of 2003 for a typical urban background location in central London and a rural background location. Ozone is present on almost all days of the year. During summer smog days, as indicated by the peaks during June–August in Fig. 4.1, elevated ozone levels are seen to exceed $140 \mu\text{g}/\text{m}^3$. The temporal correlation between rural and urban background ozone is high. In most towns and cities in northwestern Europe, there are relatively few such episode days during most years and their number shows a great deal of variability from year to year. During the winter, periods of strongly depleted ozone levels are seen during winter smog days. Again, there are relatively few of these and their number shows significant year-on-year variability.

There are no significant direct sources of ozone emission to the atmosphere and therefore all ground-level ozone has been formed in the atmosphere by sunlight-driven chemical reactions. In the troposphere, these chemical reactions involve NO_x and organic compounds as precursors from both natural and man-made sources and take place over a range of timescales from under an hour to

Fig. 4.1. Maximum daily 8-hour average ozone concentrations for each day of 2003 for an urban background location in central London (Bloomsbury, blue line) and the rural background location (Harwell, red line)



many days. Long-range transboundary transport is generally an important feature of summertime ozone episodes, with episodic peaks from regional sources superimposed on the hemispheric background.

Emissions of NO_x act as a sink for ozone as well as a source. During winter smog days, particularly under calm conditions, NO_x from motor traffic efficiently scavenges ozone and can leave its concentrations severely depleted, as shown in Fig. 4.1. This accounts for ozone levels being invariably lower in towns and cities than in the surrounding rural areas. When considering ozone in urban areas it should be remembered that in wintertime, low ozone concentrations are associated with high concentrations of other pollutants, while in summer the opposite is true and high ozone concentrations are generally associated with high concentrations of other pollutants. This pattern is illustrated by data from a typical urban background site in London (Table 4.1). Similar behaviour is anticipated in most European urban areas.

Table 4.1. Regression slopes between daily ozone^a vs daily NO_x and PM_{10} levels^b during January and July 2000 at an urban background site in London

Month	vs PM_{10} ($\mu\text{g}/\text{m}^3$ per ppb)	vs nitric oxide (ppb per ppb)	vs nitrogen dioxide (ppb per ppb)	vs NO_x (ppb per ppb)
January	-0.43	-0.56	-0.81	-3.8
July	+0.36	+0.46	—	—

^aThe daily ozone metric is the maximum 8-hour mean concentration.

^bDaily means have been used for PM_{10} , nitric oxide, nitrogen dioxide and NO_x .

Choice of ozone concentration indicator for use in health impact assessment

The Gothenburg protocol of 1999 used accumulated exposure over a threshold of 40 ppb (AOT40) as a measure to protect plants and the corresponding AOT60 to protect human health. AOT values were defined as continuous (integral) measures and calculated in practice using hourly levels.

The Joint WHO/Convention Task Force, at its seventh meeting in May 2004 (79), considered the evidence on the health effects of ozone as well as the availability and performance of models estimating ozone concentrations. Based on the evidence and models available at that time, the Task Force recommended a new index of cumulative annual exposure to ozone, to be used for the inclusion of ozone-related mortality in integrated modelling. The reasoning of the Task Force was as follows.

- There is insufficient evidence from studies of long-term exposure and mortality to allow quantification of mortality impacts. The effects on mortality of acute (daily) variations in ozone are sufficiently well-established to allow quantification.
- The principal metric for assessing the effects of daily ozone on mortality should be the maximum daily 8-hour average. This was in line with the health studies used to derive the summary estimate.
- Evidence was (and still is) insufficient to derive a level below which ozone had no effect on mortality (see Chapter 2), i.e. there is no convincing evidence of a threshold at the population level (see page 15). However, there are substantial uncertainties about the magnitude of any mortality effects at low concentrations of daily ozone, for two reasons:
 - there are uncertainties in the shape of concentration–response function at very low ozone concentrations; and
 - estimates of ozone concentrations produced by atmospheric models tend to be unreliable in the lower range of ozone concentrations.

On that basis, the Task Force recommended that, in the interests of prudence, the possible effects of daily ozone on mortality should in the first instance be quantified only when daily ozone concentrations were sufficiently high, i.e. when the maximum daily 8-hour average ozone concentration exceeded some cut-off value.

Following substantial discussion, a cut-off value of 70 $\mu\text{g}/\text{m}^3$ was recommended for integrated assessment modelling. The decision to select 70 $\mu\text{g}/\text{m}^3$ was based on two arguments:

- a statistically significant increase in mortality risk estimates was observed at ozone concentrations above 50–70 $\mu\text{g}/\text{m}^3$; and
- according to the opinion of experts present at the seventh meeting of the Task

Force, more reliable estimates from atmospheric models were available for concentrations above $70 \mu\text{g}/\text{m}^3$.

In implementing the cut-off, no effects of ozone on health would be calculated on days with a maximum daily 8-hour average below $70 \mu\text{g}/\text{m}^3$. For days with ozone concentrations above $70 \mu\text{g}/\text{m}^3$ as a maximum daily 8-hour average, only the increment exceeding $70 \mu\text{g}/\text{m}^3$ would be used to calculate effects.

The effects accumulated over a certain period (e.g. a year) are the sum of impact estimates for each of the days in the period. Owing to the linearity of the concentration–response curve, the accumulated impact estimate is proportional to the sum of concentrations over the cut-off. Effectively, it meant that the exposure parameter was the sum of excess of maximum daily 8-hour averages over the cut-off of $70 \mu\text{g}/\text{m}^3$ calculated for all days in a year. The term SOMO35 (Sum Of Means Over 35 ppb) was proposed as a name for this indicator of cumulative annual exposure (see Box 4.2 for a more detailed description).

The Task Force also made the following recommendations.

- It was highly likely that the overall effects of ozone were underestimated by this approach. Therefore, a sensitivity analysis applying no cut-off (= SOMO0) should be made. This estimate would indicate an upper estimate of the attributable effects of ozone on mortality. The same coefficient would be used.
- Ozone effects should be assessed over the full year.
- For assessing ozone exposure in urban areas, urban background concentrations should be used. To be in line with most of the evidential health studies, it was regarded as sufficient to use one average ozone concentration per city.
- At this stage, there were insufficient data to distinguish (susceptible) subpopulations and the calculations should be applied to total population.

These recommendations were accepted by the 23rd session of the Working Group on Effects and used by IIASA for modelling the impacts of ozone in the RAINS model, and so were used in the health impact assessment of CAFE.

SOMO35 is an important index in that it reflects the Task Force's assessments of the evidence at a point in time. It was therefore used in major quantification studies in Europe, including CAFE. It should not, however, be seen as a new, universal or lasting index. In particular, it reflects the evidence base as it was in 2004, i.e. that (a) effects from studies of long-term exposure were not well enough established to be quantified; and (b) there were substantial uncertainties about the slope of the concentration–response function at lower concentrations, say below $70 \mu\text{g}/\text{m}^3$. The SOMO35 index should be reconsidered if and when that evidence base changes to an important degree.

The distribution of SOMO35 over the population living in cities where at least one (sub)urban background ozone monitoring station is operational is given in

Box 4.2. What is SOMO35?

SOMO35 is an indicator of the accumulated ozone concentration in excess of 35 ppb (70 $\mu\text{g}/\text{m}^3$). It was developed for use in health impact assessment, as explained on pages 33–35. This indicator is defined as:

$$\text{SOMO35}_{\text{uncorrected}} = \sum_i \max\{0, C_i - 35 \text{ ppm}\}$$

where C_i is the maximum daily 8-hour average concentration and the summation is from day $i=1$ to 365 per year. SOMO35 has a dimension of ppb \cdot days if 35 ppb is used and $\mu\text{g}/\text{m}^3 \cdot$ days if 70 $\mu\text{g}/\text{m}^3$ is used in the equation. SOMO35 is sensitive to missing values (i.e. days when maximum daily 8-hour average concentrations are unavailable); where such daily data are missing a correction to full-time (e.g. annual) coverage is needed. This is done here by introducing:

$$\text{SOMO35} = \text{SOMO35}_{\text{uncorrected}} \cdot \frac{365}{N_{\text{valid}}}$$

where N_{valid} is the number of valid daily values. Corrected values of SOMO35 are calculated and used when sufficient valid daily measurements are available. For practical reasons, a data coverage of at least 75% is required (i.e. $N_{\text{valid}} > 273$) and the days with missing data should not concentrate in one season.

The figure below shows time-series of maximum daily 8-hour average concentrations measured in one of the urban background sites in Austria in 2003. The blue area above the 70- $\mu\text{g}/\text{m}^3$ level corresponds to the SOMO35 value. It is clear that the largest contributions to SOMO35 are made during the summer months, although the figure also shows contributions during the colder months.

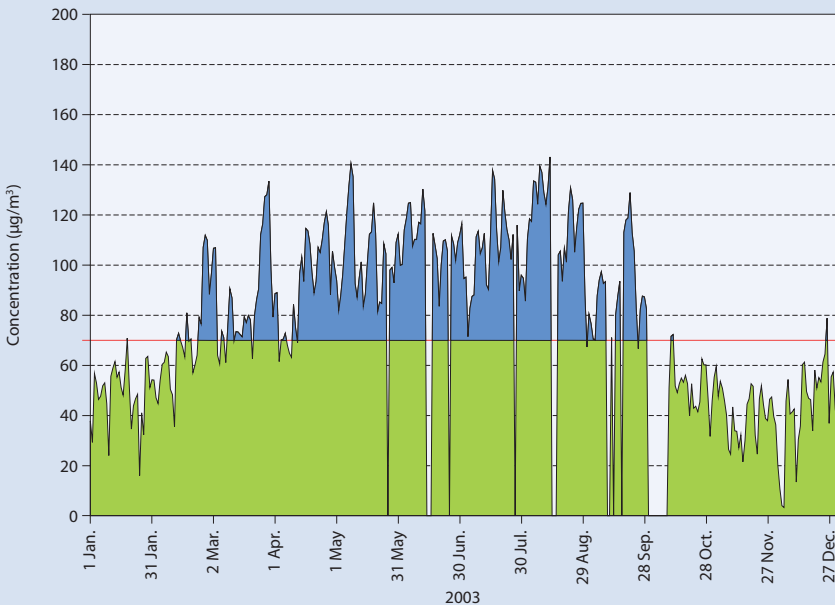
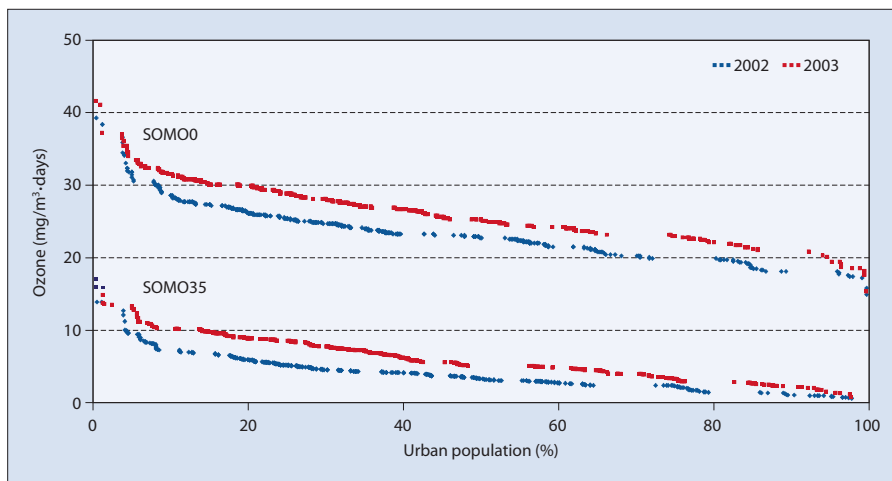


Fig. 4.2. Cumulative distribution of SOMO35 and SOMO0 values over the population living in cities with at least one operational (sub)urban background monitoring station, 2002 and 2003



Source: AirBase, © EEA, Copenhagen, 2008 (80).

Fig. 4.2 for the situation in 2002 and 2003. Comparison with aggregated maximum 8-hour levels without a threshold (SOMO0) shows that approximately two thirds of the accumulated exposures occur at levels below $70 \mu\text{g}/\text{m}^3$ (35 ppb). SOMO35 in urban areas tends to increase towards the southeastern parts of Europe (see Fig. 4.3).

Although a comparison between regions is limited owing to differences in the spatial coverage of the monitoring, clear differences between the regions can be noted. The highest values are observed in southern Europe, with a population-averaged mean of $6700 \mu\text{g}/\text{m}^3 \cdot \text{days}$, and in central and eastern Europe ($3930 \mu\text{g}/\text{m}^3 \cdot \text{days}$). The lowest values are in the northwestern region ($1730 \mu\text{g}/\text{m}^3 \cdot \text{days}$).

Table 4.2. Fractions of populations exposed to ozone levels exceeding the EU directive target value of $120 \mu\text{g}/\text{m}^3$ for more than 25 days a year, by region

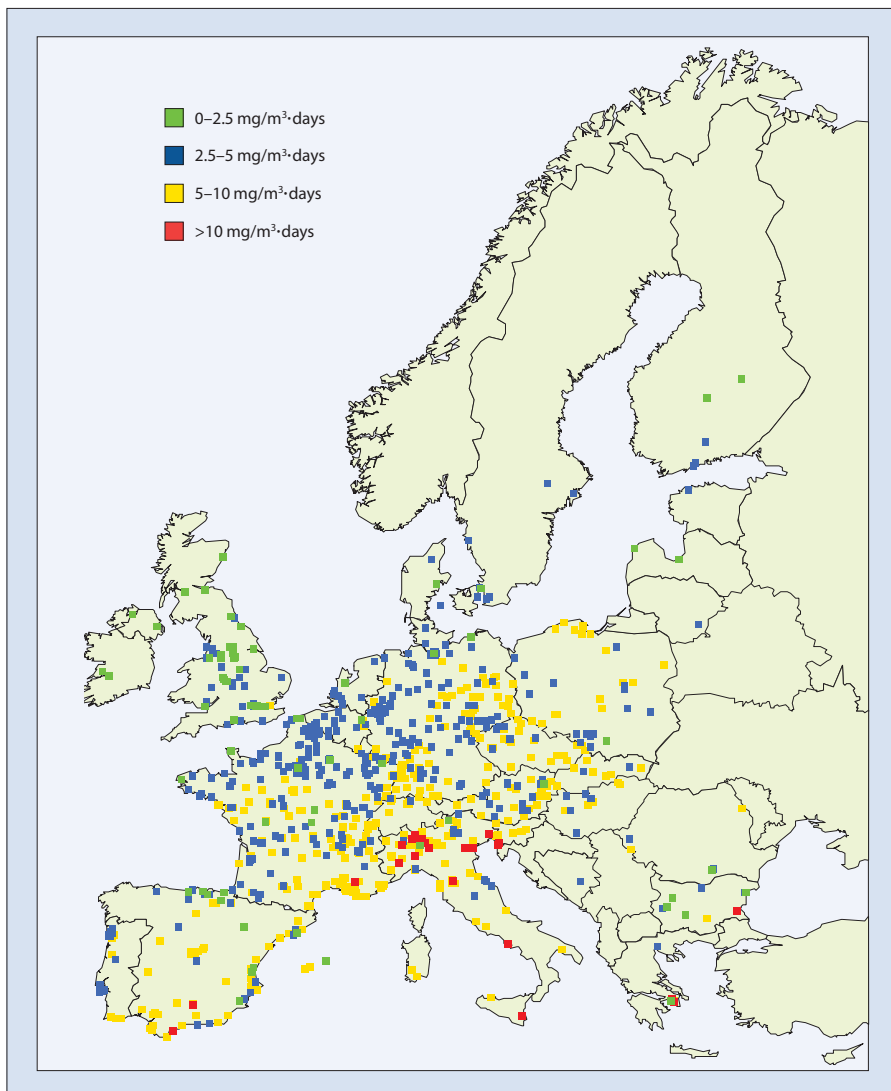
Region ^a	2002	2003
Northern Europe	0%	0%
Northwestern Europe	0–10%	40–50%
Central and eastern Europe	20–30%	80–90%
Southern Europe	60–70%	60–70%

^a Northern Europe: Denmark, Estonia, Finland, Iceland, Latvia, Lithuania, Norway, Sweden; Northwestern Europe: Belgium, France (north of 45°N), Ireland, Luxembourg, Netherlands, United Kingdom; Central and eastern Europe: Austria, Czech Republic, Germany, Hungary, Poland, Slovakia, Switzerland; Southern Europe: Cyprus, France (south of 45°N), Greece, Italy, Malta, Portugal, Slovenia, Spain.

Source: AirBase (80).

The population-weighted mean for northern Europe is $2860 \mu\text{g}/\text{m}^3 \cdot \text{days}$.

In the EU directive on ozone (77), a target value for the protection of human health is set for 2010 and defined as a maximum daily 8-hour average of $120 \mu\text{g}/\text{m}^3$, not to be exceeded on more than 25 days per calendar year averaged over three years. In 2002, a relatively low-ozone year, 20–30% of the urban population was exposed to concen-

Fig. 4.3. SOMO35 values measured at (sub)urban background stations in 2006

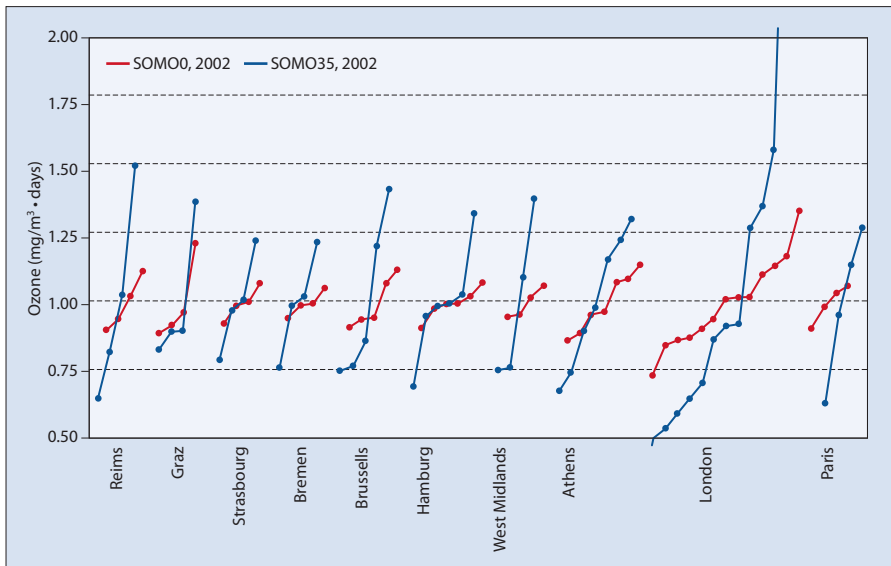
Source: AirBase, © EEA, Copenhagen, 2008 (80).

trations above the target level on more than 25 days; in 2003, a high-ozone year, this increased to about 60%. Exceedances were more frequently observed in the southern and central parts of Europe (Table 4.2). The geographical differences in ozone values measured with SOMO35 are less pronounced than is the case for the number of days when the EU target value is exceeded.

Local-scale variations in ozone concentration

Variations in concentrations within a city were examined using the information available in AirBase (80). Cities were selected with at least four (sub)urban

Fig. 4.4. Variation in ozone levels expressed as SOMO35 or SOMO0 at different monitoring stations within urban areas relative to the urban mean values for each city



Source: AirBase, © EEA, Copenhagen, 2008 (80).

background stations. After calculating the average city concentration, the ratio of ozone levels at each individual station with the average value was calculated (Fig. 4.4). With the exception of London, local variations within a city were about $\pm 30\%$. In Fig. 4.4, where the cities are ranked according to their size, local variations seem not to be a function of city size or location. This indicates that SOMO35 for each site depends strongly on local conditions. SOMO0 is a less variable parameter, local urban variation being reduced to about $\pm 10\%$.

Observed ozone trends

Ozone is a secondary pollutant whose production is strongly influenced by meteorological conditions from year to year in addition to anthropogenic emissions. Thus trends in surface ozone due to changes in emissions may be masked by the varying meteorology (81,82). Nevertheless, it seems that peak ozone values declined in many regions of Europe during the 1990s, whereas long-term average and background values were seen to increase at many sites. A more thorough review of these topics, covering measurement data from different regions of Europe, can be found in Lövblad et al. (83).

Global trends

Surface ozone concentrations in background locations have been estimated to have increased 2–3-fold during the last century, and levels at the beginning of the twentieth century may have been around $20 \mu\text{g}/\text{m}^3$ (84,85). There are many problems associated with these early measurements, however, and pre-industrial

ozone levels may have been closer to $40 \mu\text{g}/\text{m}^3$, as measured in Switzerland in the 1950s (86). Model calculations simulating the same period typically give ozone concentrations of around $40 \mu\text{g}/\text{m}^3$ (87,88).

Studies of ozone sonde data in the free troposphere point to a general increase in free tropospheric ozone up to the mid-1980s, followed by a mixed picture with many sites/regions showing no significant or even a downward trend (89–91). However, analyses of the clean wind sector at several rural sites indicate an increase in background ozone levels after the mid-1980s (92), particularly in winter. Contrary to the ozone sonde data, model calculations indicate that the increase in free tropospheric ozone continued after the mid-1980s (88,93). Chen et al. (94) and Wu & Chan (95) report increasing ozone trends in China (Taiwan and Hong Kong Special Administrative Region, respectively) during the 1990s and the early 2000s.

Hemispheric changes in ozone air quality have affected ozone distribution across northwestern Europe. For most urban pollutants, the location of major population and industrial centres on the western (upwind) side of Europe has been an air quality advantage because clean Atlantic Ocean air masses generally bring low pollution levels. However, ozone production from both natural and man-made ozone precursors on the hemispheric scale ensures that these otherwise clean air masses always contain some background or baseline levels of ozone. This is why ozone is almost invariably present in most towns and cities across Europe on almost all days. From 1987 to 1997, background ozone levels at Mace Head, Ireland increased at about 0.14 ppb per year, whereas since 2000, background levels have remained constant and have if anything declined slightly (96). Winter and spring levels have been rising somewhat faster than summer levels. A similar trend has been observed in clean Pacific Ocean air masses entering the North American continent, so policy-makers can be sure that it is, indeed, a hemispheric-scale phenomenon (97).

Observed ground-level ozone trends in Europe

The distributions of ozone concentrations observed at any site, during any year, have been changing throughout the period over which monitoring has been carried out. Some years show a greater frequency and intensity of summertime ozone episodes and wintertime depletion events than others.

Assessment of ozone levels in rural areas is mainly based on results from the EMEP network. In the United Kingdom, it is estimated that peak ozone concentrations at the EMEP background stations declined by about 30% in the period 1986–1999 (98). At the same time, there was a slight increase in the annual average concentration. Based on observed trends in individual VOC concentrations, together with detailed model calculations of the potential for ozone formation for individual VOCs, Derwent et al. (99) concluded that the downward trend in episodic peak ozone concentrations in northwestern Europe should have been

about 4.4–5.3 $\mu\text{g}/\text{m}^3$ per year during the 1990s, to which a further 1.4 $\mu\text{g}/\text{m}^3$ per year should be added from the non-monitored VOC species. When trends in other anthropogenic emissions (NO_x , sulfur dioxide and carbon monoxide) were added together, a net downward trend of 6.8 $\mu\text{g}/\text{m}^3$ per year was estimated, which compares closely with the observed reductions in ozone of 3.8–5.8 $\mu\text{g}/\text{m}^3$ per year across the British Isles.

An evaluation for the Nordic countries (100) indicated that the frequency of high ozone values decreased in the 1990s. Several results taken together indicate that it is likely that peak values have fallen in southern Norway and southern Sweden owing to reduced European emissions during the 1990s. The model results indicate a reduction of the order of 30 $\mu\text{g}/\text{m}^3$ for the highest ozone peak values, with lower values for the less pronounced episodes over the decade. The 99th percentiles of summer six-month hourly data have probably been reduced by the order of 10–20 $\mu\text{g}/\text{m}^3$ in the same region. For Finland and for the northern part of the region, the conclusions are more difficult to assess and become more uncertain. Although the model indicates similar results for southern Finland as for southern Norway and Sweden, the agreement with the measurements is poorer. This possibly reflects the fact that ozone episodes in Finland in general are more linked to transport from the east than from Norway and Sweden, and that emission data for the Russian Federation and eastern Europe are generally less well-established.

Data from selected EMEP rural background sites do not show consistent trends in peak values throughout Europe in 1990–2006. A slight decline in peak values was seen in EMEP sites in Italy and the Netherlands, but at other EMEP stations in Europe there was no clear decrease or increase in peak levels (Fig. 4.5).

There have been three opposing influences on rural ozone levels in Europe over the period 1990–2006:

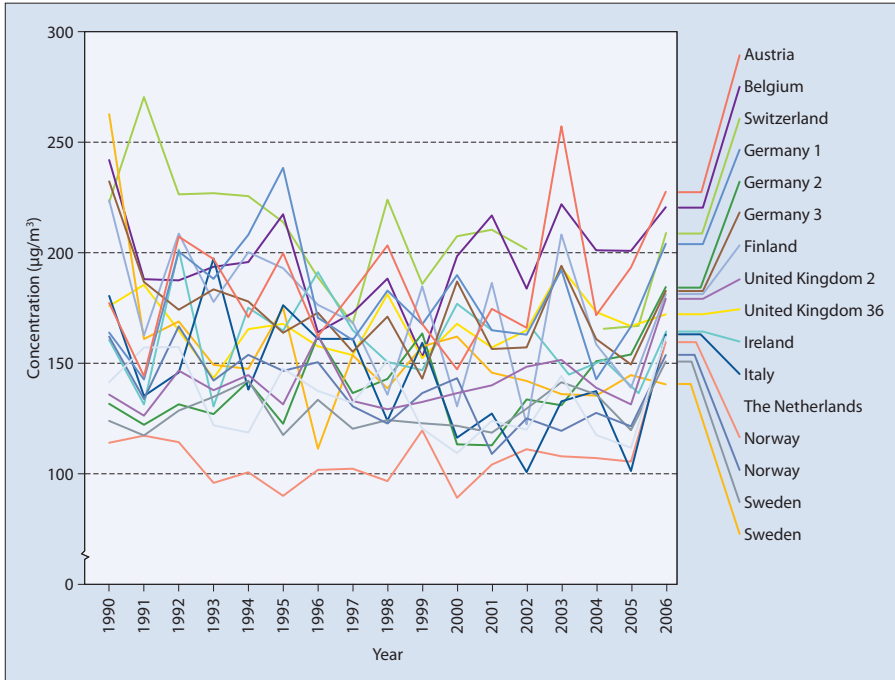
- the decreasing intensity of the regional ozone pollution episodes, tending to reduce the ozone metric;
- the decreasing depletion of ozone by NO_x emissions from traffic and other sources; and
- the growth in hemispheric or global baseline ozone.

Further, long-term changes in climatic conditions could lead to a still unquantified positive or negative change in ozone concentrations.

Overall, an approximate balance was maintained between these influences over the period at rural EMEP sites.

Urban ozone levels are most affected by changing patterns of emissions from traffic. Three-way exhaust gas catalysts have reduced the extent of ozone scavenging by NO_x emissions from petrol engines. As a result, ozone levels in towns and cities have begun to rise back towards the levels found in the surrounding

Fig. 4.5. Time-series of the maximum daily 8-hour average ozone concentrations monitored at a selection of long-running rural EMEP background sites between 1990 and 2006



Source: Fjæraa & Hjellbrekke (101).

countryside. Ozone depletion events during the winter have become less severe. There has been a tendency, therefore, for levels to rise during much of the year while episodic peak levels during the summer have fallen.

To illustrate trends in ozone levels observed in the last decade, Fig. 4.6 presents the frequency distributions of the 8760 hourly ozone average concentrations measured at an urban background site in central London during 1991 and 1998. There was a marked shift in the frequency distribution of ozone concentrations over the period, bringing a much lower frequency of low ozone concentrations ($<20 \mu\text{g}/\text{m}^3$) and a much higher frequency of ozone concentrations in the $20\text{--}80 \mu\text{g}/\text{m}^3$ range. This is likely to be due mainly to reduced NO_x emissions but will also reflect the steadily increasing ozone background, especially during the winter. Similar behaviour is anticipated in most towns and cities in northwestern Europe.

According to AirBase data, observed ozone trends are in general not statistically significant in the period 1996–2002, indicating a need for more in-depth analysis, including accounting for meteorological variability. A falling trend in the peak values observed earlier levelled off during the 1990s and the median concentrations show an increasing trend for all station types. The increase was

Fig. 4.6. Frequency distributions of the hourly ozone concentrations measured during 1991 and 1998 at Bridge Place, a typical urban background site in central London

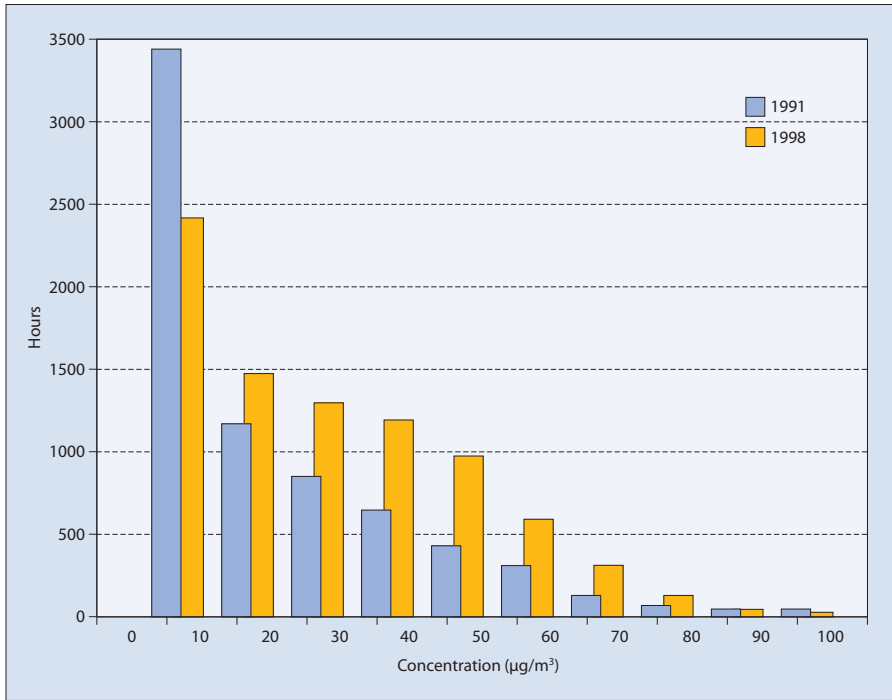
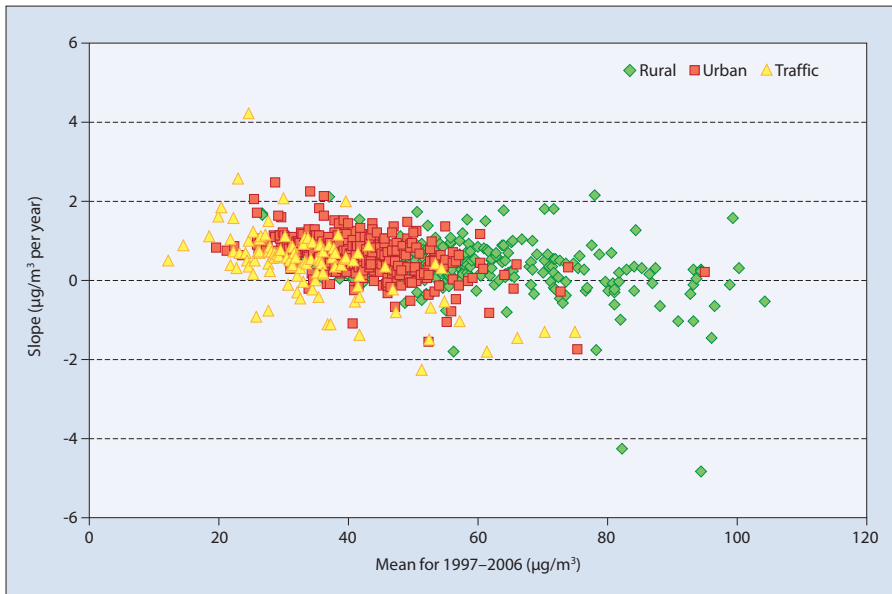


Fig. 4.7. Change in annual mean concentration plotted against the mean value over the period 1997–2006



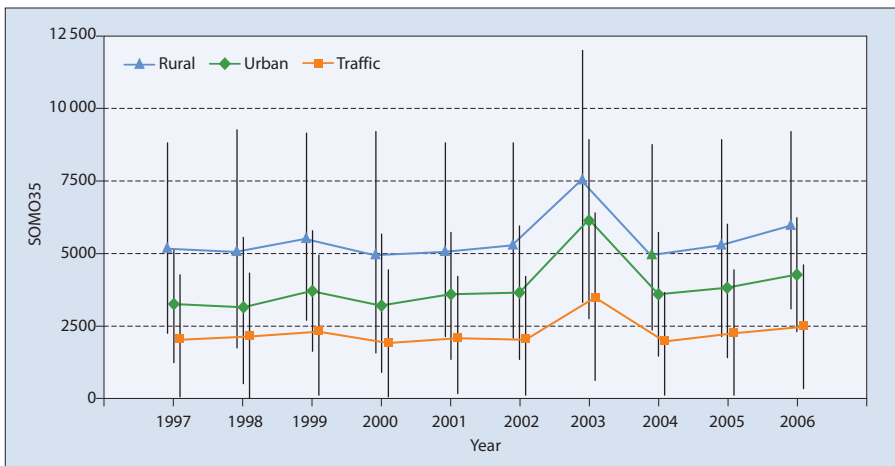
Source: AirBase, © EEA, Copenhagen, 2008 (80).

more pronounced, however, for street and other urban stations, which represent exposures of the largest populations in Europe (102). Fig. 4.7 shows the change in annual mean ozone concentrations in Europe from 1997 to 2003 as a function of the mean value during this period. The figure shows that there is a tendency towards increasing annual mean ozone values at all station types, although the trend is not statistically significant at all stations. No correction for meteorological variability has been applied; some bias towards positive tendencies might have been introduced owing to the extreme values observed in 2003.

For the urban background, and especially for the street stations, there is a slight predominance towards an upward trend in annual mean concentration, which fits with the effect of decreasing urban and traffic NO_x concentrations (76). Trends in annual mean concentrations are statistically significant at the 0.10 level only at about 30% of the stations. Three quarters of all stations have a linear trend slope, with a coefficient within $\pm 1 \mu\text{g}/\text{m}^3$ change in annual mean per year. These tendencies are, for the average of all stations, quite similar in three regions (northwestern, central/eastern and southern Europe); on average, a positive slope of $0.4\text{--}0.5 \mu\text{g}/\text{m}^3$ per year is observed. Assessment of the EMEP ozone data (83) shows a slightly larger trend ($0.6\text{--}1.0 \mu\text{g}/\text{m}^3$ per year) for rural stations in the north and west of Europe. In northern Europe, a zero slope is observed. It has to be stressed, however, that the number of stations and the distribution over the three station types differ widely among the regions. This seriously hampers comparison between regions.

The annual variations in SOMO35 show an almost unchanged averaged level since 1997, with increased levels in 2003 and a slightly increasing trend in

Fig. 4.8. Annual variations in SOMO35 values, averaged over all stations that reported data over at least six years in the period 1997–2006



Note: The vertical bars indicate the 10th and 90th percentiles.

Source: AirBase, © EEA, Copenhagen, 2008 (80).

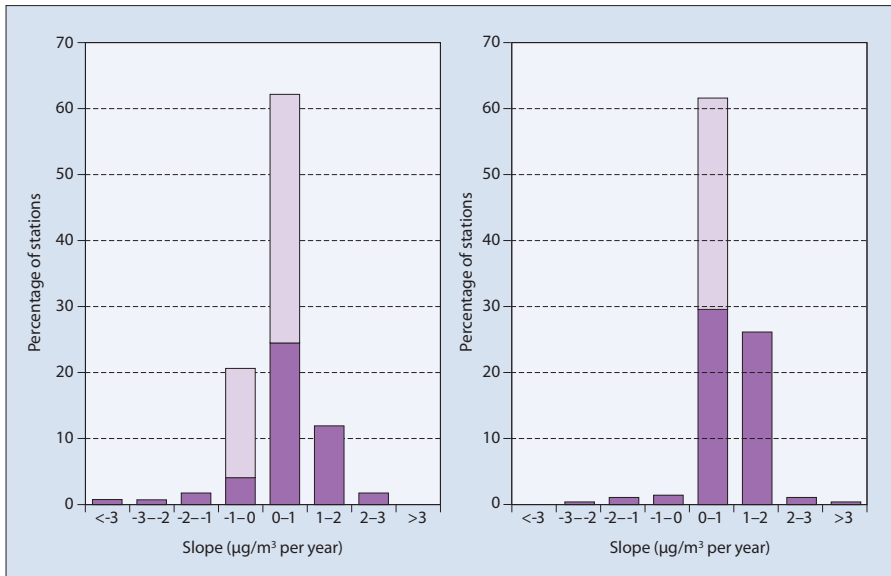
2005–2006 compared with the period before 2003 for all three station types (rural, (sub)urban background and traffic hot spot) (Fig. 4.8). Averaged over all stations, a positive tendency is observed in the central/eastern region and negative tendencies in the other regions. A significant trend in SOMO35 is found at one out of every four stations.

In view of the importance of urban exposure levels, a clear understanding of urban ozone trends is required. Urban ozone trends have been examined for (sub)urban background sites over the period 1997–2003 and the results are summarized in Fig. 4.9. Upward trends were found at the vast majority of the urban sites (266 out of 290) and downward trends at only 24 sites. A total of 180 sites showed positive trends that are statistically significant (at a 10% level of significance). Rural stations also showed upward though slightly less strong trends.

The strong upward trends in the ozone metric observed at almost all of the urban sites are caused by the diminution of ozone depletion by chemical reactions with NO_x, which has been caused, in turn, by the reduction in emissions from petrol-engine vehicles. According to this analysis, the upward trends in daily concentrations occur in spite of a decrease in maximum levels of ozone during regional ozone pollution episodes.

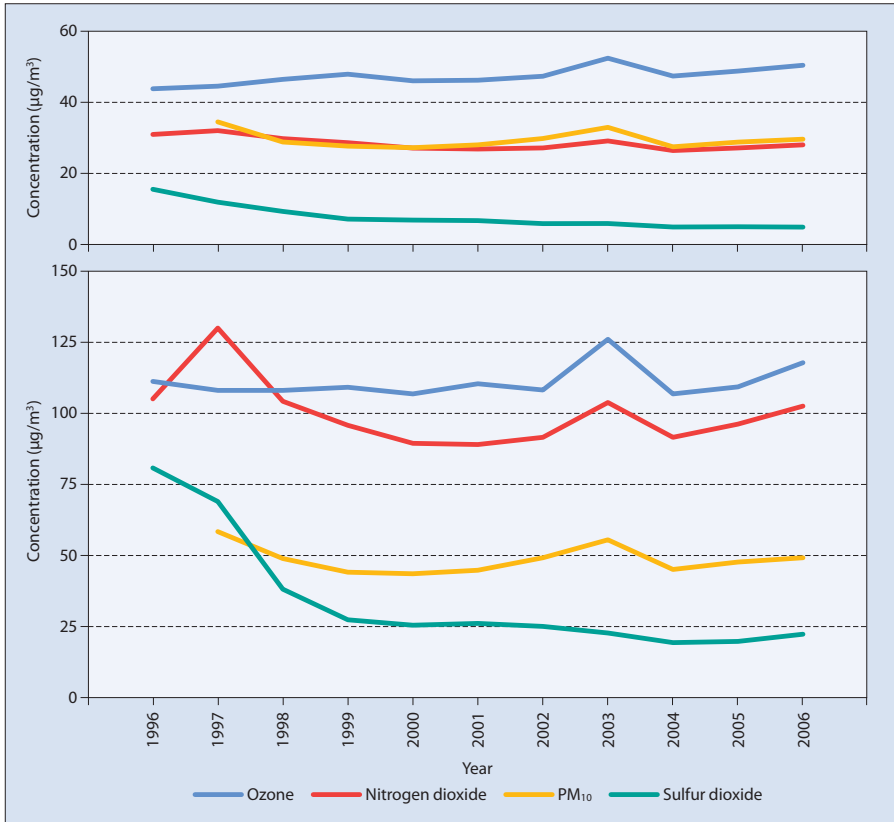
In Fig. 4.10, trends in ozone concentrations over the last several years are compared to tendencies and trends for other pollutants, as an average for all types of station. For each year, the plotted value represents the average of all stations in all

Fig. 4.9. Annual average maximum daily 8-hour average ozone concentrations observed at rural (left) and (sub)urban (right) background stations, 1997–2006



Note: The darker colour bars refer to stations having a statistically significant trend at a 10% level; the lighter colour bars refer to stations having a non-significant trend.
 Source: AirBase, © EEA, Copenhagen, 2008 (80).

Fig. 4.10. Measured concentrations of sulfur dioxide, nitrogen dioxide, PM₁₀ and ozone in Europe, as an average of all stations, 1996–2006



Note: upper panel, annual means; lower panel, 26th highest daily value.

Source: AirBase, © EEA, Copenhagen, 2008 (80).

countries whose data are available in AirBase (only stations with data covering at least 75% of the period are included).

While ozone concentrations (both annual means and daily values) tend to increase, the average annual mean for sulfur dioxide shows a strong downward trend until about 2000, followed by a flattening out (and even a small increase in the last year). Nitrogen dioxide and PM₁₀ also show a downward trend (except for peaks in the high-percentile short-term level in 1997 and 2003), but the relative as well as absolute concentration reduction for these pollutants is much less than for sulfur dioxide.

For ozone, nitrogen dioxide and PM₁₀, there was an increase from 2001 to 2003 in the high-percentile short-term concentrations. This may indicate that meteorological conditions could explain the increase.³

³ Note that PM₁₀ data as stored in AirBase are used here; some “PM₁₀ trends” may be caused by an inconsistent use of correction factors over the years.

Modelling techniques and validation

Modelling of ozone levels is required in order to improve spatial coverage in data-sparse areas, as well for estimating future trends in ozone levels and formulating policies to control adverse effects on vegetation and human health. The Unified EMEP photochemistry model (82,103) has been developed as a tool for calculating long-term source–receptor relationships of air pollutants on a regional scale in Europe as part of the EMEP programme. The model describes the transport, chemical transformation and deposition of approximately 75 species, among them ozone, carbon monoxide, NO_x, sulfur dioxide, ammonia, numerous VOCs, nitric acid, and primary fine and coarse PM.

The model domain is centred over Europe and also includes most of the North Atlantic and the polar region, with a horizontal resolution of approximately 50 × 50 km and 20 vertical layers below 100 hPa. Dry deposition is modelled using a resistance approach. Wet scavenging is set to be proportional to the precipitation intensity, using species-specific scavenging coefficients. Photolysis rates are tabulated for clear sky and cloudy conditions. Boundary conditions of free tropospheric ozone and other components are derived mainly from observed climatic conditions. A detailed model description can be found in Simpson et al. (103) and Fagerli et al. (104).

Modelling of local, small-scale effects

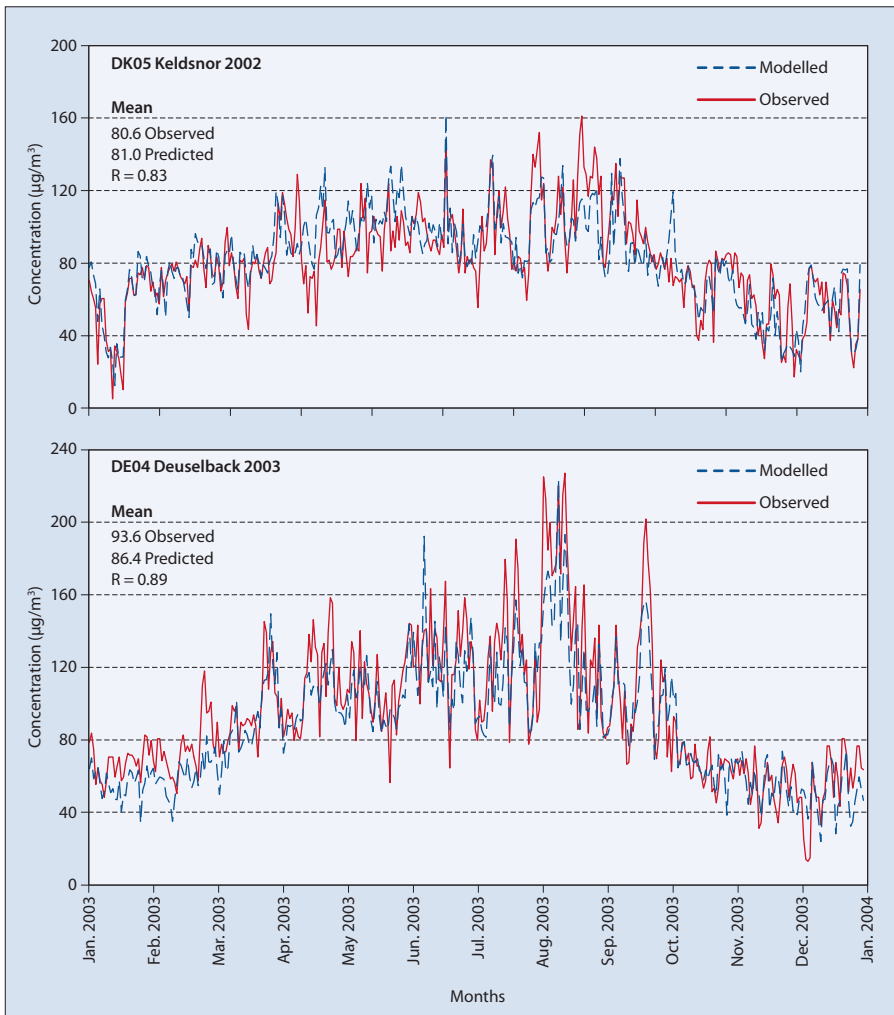
The regional ozone levels that are the focus of EMEP can be locally modified by other air pollutants emitted around urban areas. A common modification is the local depression in ozone levels in the NO_x plume within and downwind of the city. Emissions from city areas contribute to ozone formation further downwind, and such city-plume enhancements are expected to be most important in warm weather, especially in southern Europe, but these effects take tens to hundreds of kilometres to develop. Local meteorological conditions can also play a strong role in determining the ozone concentration observed around cities. Sea breezes have been studied extensively in this context (105), but many types of local (meso-scale) circulation are associated with elevated ozone levels (see, for example, Louka et al. (106) for further details and many references).

Studies of ozone around urban areas have increased in recent years. Examples are the ESQUIF project around Paris (107), the BERLIOZ project around Berlin (108), BOCCALINO and PIPAPO around Milan and southern Switzerland (109,110) and the ESCOMPTE project (111). Clear instances have been found in these studies of ozone plumes downwind of major cities; for example, Domen et al. (112) observed that the ozone level in the plume downwind of Milan was around 40–80 µg/m³ higher than in adjacent areas. However, even in regions where local ozone production may be expected to be strong, the regional ozone concentrations often dominate. This was seen in the Heilbronn experiment in southern Germany, where strict emission reductions within the city had little

effect on ozone concentrations even though they reduced the nitrogen dioxide level considerably (113).

Moussiopoulos et al. (114) investigated the relationship between regional-scale emission controls, calculated with the Lagrangian EMEP ozone model, and local controls, calculated with the simple OFIS model, for two cities, Stuttgart in Germany and Athens in Greece. They concluded that controlling urban VOC levels is effective in reducing ozone primarily on the local or urban scale, whereas controlling urban NO_x levels may increase urban peak ozone while contributing to an effective reduction of regional ozone.

Fig. 4.11. Modelled results from the EMEP model compared with observed ozone concentrations (maximum daily 1-hour average values) at sites in Denmark (above) and Germany (below)



Source: Simpson et al. (117).

Comparison with measurements

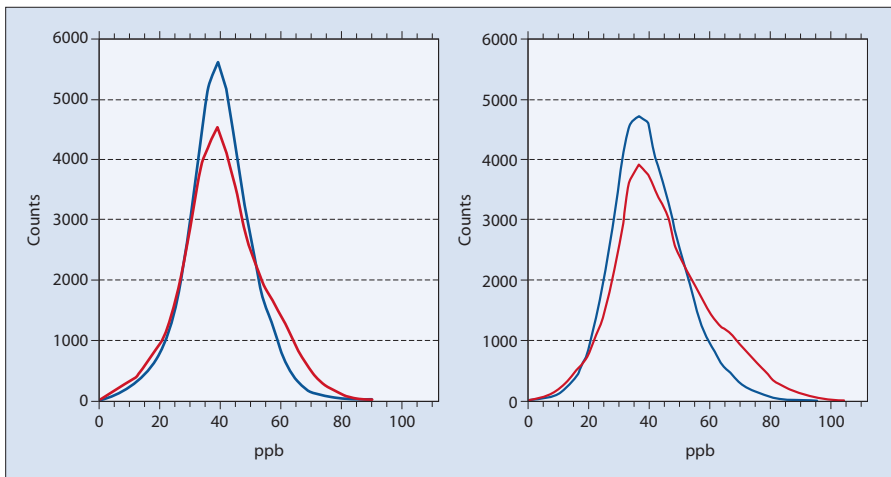
The EMEP model has been extensively tested (115–118) and reviewed (119,120). Earlier versions of the model have participated in several model comparison studies (121,122).

Fig.4.11 illustrates the performance of the EMEP model, comparing maximum daily ozone values over the year at sites in Denmark and Germany. Typical features of these plots are the good reproduction of the seasonal cycles and the capture of most, though not all, episodes. There is a tendency to under-prediction of ozone for the highest concentrations and over-prediction at the minima. Problems in reproducing the extreme ozone levels may be linked to the model resolution, as the measurement sites are likely to pick up plumes of ozone formation or depletion downwind of sources that are not fully resolved in the model. Many more time-series of this type, covering the whole EMEP network and many years of simulations, can be found in Simpson et al. (116,117) and Jonson et al. (118).

The maximum daily ozone concentrations from the model and measurements can also be displayed as frequency distributions. Fig. 4.12 shows the frequency distribution of measured and modelled maximum daily ozone from 127 sites measuring ozone in 2002 and 2003. As in the time-series plots above, it is seen that the EMEP model tends to under-predict the number of very high and very low values measured, although the distribution is captured quite well as a whole.

Fig. 4.13 shows the scatter plot of EMEP modelled vs observed SOMO35 for the years 2002 and 2003. As noted earlier, SOMO35 is a rather sensitive index of ozone exposure, so as expected the scatter is large, but the model does capture the

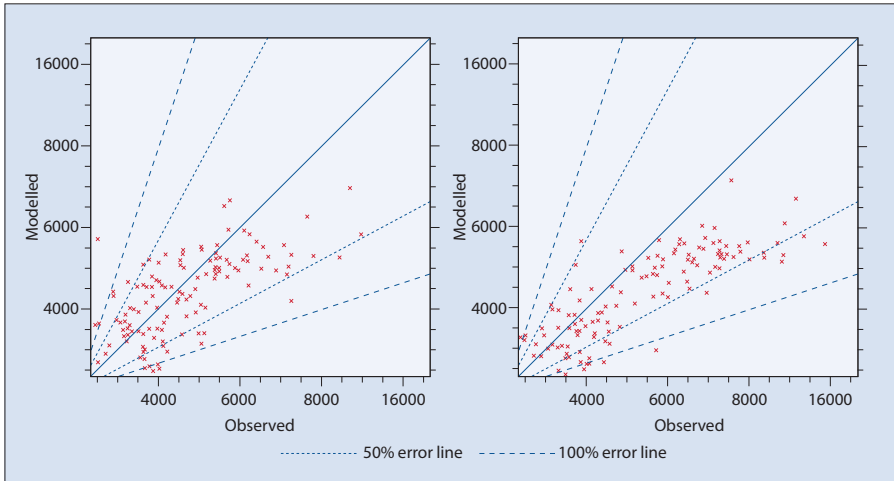
Fig. 4.12. Frequency distributions of modelled (blue line) versus observed (red line) ozone values in 2002 (left) and 2003 (right), derived from all daily maximum ozone values over 127 stations



Source: Simpson et al. (117)

variations in SOMO35 quite well. For 2002, the model shows a slight tendency to over-predict SOMO35, whereas for 2003, there is a clear tendency for the model to under-predict SOMO35 values. For both years the great majority of sites lie within the 50% error lines.

Fig. 4.13. Comparison of modelled vs observed SOMO35 values ($\mu\text{g}/\text{m}^3\cdot\text{days}$) for 2002 (left) and 2003 (right)



Source: Simpson et al. (117).

Ozone and climate change

The relationship between ozone and climate change is dynamic. Ozone, especially stratospheric ozone, is a greenhouse gas influencing the climate. It has been estimated that the past increase in the tropospheric abundance of ozone may have provided the third largest increase in direct radiative forcing since the pre-industrial era (123,124).

The most significant meteorological variables directly affected by climate change are temperature and specific humidity. Climate models suggest that the relative humidity of the atmosphere will remain roughly constant in future and, since a warmer atmosphere can hold more water vapour, this implies that the specific humidity will increase. Higher temperatures can increase the reaction rates producing ozone and reduce the amount of NO_x . Increased water vapour will increase the decomposition of ozone. Throughout most of the troposphere, the water vapour effect leads to a net reduction in ozone (125), but close to the surface over polluted regions some climate change models project an increase in ozone pollution (126) owing to a decrease in peroxyacetyl nitrate (PAN) formation. Emissions of VOCs from biogenic sources (mainly forests) will also be affected, by direct changes in temperature and other meteorological factors and by changing carbon dioxide levels and growing seasons. There are great uncer-

tainties, however, surrounding the net effects of these changes on VOC emissions and ozone formation.

While water vapour and temperature increases have opposite effects on ozone, they both increase the oxidizing capacity of the atmosphere by increasing production of the hydroxyl radical (OH). In a warmer, wetter climate, air pollutants will therefore be oxidized faster and the concentrations of primary pollutants (such as NO_x and sulfur dioxide) near to sources will decrease. Simultaneously, the concentrations of secondary pollutants (such as nitric and sulfuric acids) near the source will increase. The oxidized pollutants tend to be more soluble than the primary pollutants, and hence more easily removed. On a global scale, the projected increase in oxidizing capacity will tend to reduce the abundance of most pollutants in the atmosphere, although some specific chemical species may increase. Some of the predicted climate changes are robust (temperature, water vapour) while others vary considerably between models (precipitation, soil moisture). However, year-on-year changes in ozone levels have been seen to be as large as the overall emission/climate-related changes caused before 2030, indicating that cause and effect remain difficult to identify on this timescale. These effects have been studied in several recent modelling exercises.

Langner et al. (127) simulated the effects of a 2.6 °C increase in temperature and found decreases in nitrate and sulfate deposition over central Europe due to decreases in precipitation. They also found less removal of total reactive nitrogen oxide (NO_y) species by precipitation, leading to a greater abundance of surface ozone. Zeng & Pyle (128) found less sensitivity to water vapour changes and more sensitivity to climate-change-induced increases in the mid-latitude stratosphere-troposphere exchange of ozone, associated with higher ozone concentrations in the lower stratosphere resulting from colder temperatures. The contrasts in sensitivity are partly related to the resolution and parameterization of stratospheric processes.

Stevenson et al. (129) described the results of a comparison of 26 differently formulated chemical transport models (meteorology not influenced by the chemical fields) and climate change models (whereby the chemical fields enter the radiation calculations of the driving general circulation model and directly influence the dynamics) organized under the auspices of ACCENT (130), the results of which fed into the Fourth Assessment Report from the Intergovernmental Panel on Climate Change (IPCC) (131). These experiments are based on new “optimistic”, “likely” and “pessimistic” IIASA emission scenarios. Ten of these models are also used to simulate the effect of climate change on atmospheric chemistry between 2000 and 2030. Such a study helps to quantify the uncertainties associated with future projections. The average estimates from the ten models indicate a decrease in ozone concentration by 0–2 µg/m³ in most parts of the world except over the continental parts of Europe and Asia, where an increase of 1–3 µg/m³ is expected. However, the addition of one standard deviation to the

average estimates changes the picture from a global decrease to almost a global increase in levels.

Bell et al. (132) investigated how climate change could affect ambient ozone concentrations, using an hourly concentration model for 50 United States cities for 1990 and 2050. Future concentrations were based on the IPCC A2 scenario and the impact of altered climate on ozone was estimated. The maximum 1-hour ozone levels were estimated to increase on average by almost $10 \mu\text{g}/\text{m}^3$ (maximum $19.2 \mu\text{g}/\text{m}^3$), the highest increases occurring in cities with current high pollution levels.

5. Population exposure projections

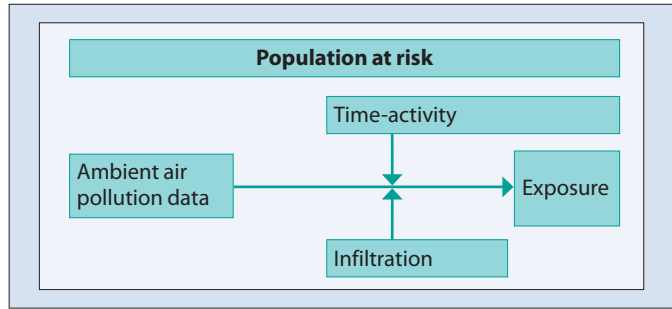
KEY MESSAGES

- The highest levels of exposure to ozone are estimated for southern Europe, with the highest levels found in northern Italy.
- Regional differences in exposure levels across Europe are expected to diminish in the next decade. Exposures in continental Europe are projected to fall by 20–30% in southern France, Germany, northern Italy and Switzerland and to rise in the United Kingdom and Scandinavia.
- It can be expected that population exposure will increase regardless of currently planned precursor emission reductions, owing to increasing background levels and reduced ozone depletion in urban areas.
- Ambient concentrations are poor estimates of personal exposure. Ozone exposure during winter may be reduced owing to the fact that more time is spent indoors. Building structures and limited ventilation affect ozone penetration to the indoor environment, especially during winter but also during summer.

Personal exposure measurements are not well-correlated with ambient fixed-site measurements. To account for this, in some studies, additional information (such as activity patterns) has been used to improve personal exposure estimates based on fixed-site measurements. Since ozone is a highly reactive gas, concentrations indoors are generally less than 50% of those in ambient air because ozone is removed by indoor surfaces as well as by gas-phase reactions (133). Most homes have very few indoor sources, which are typically such machines as copiers and electrostatic air cleaners. Outdoor ozone levels vary across city areas because ozone is scavenged in the presence of NO_x. Early-morning and late-night exposures outdoors are lower because of the diurnal cycle of ambient ozone. Thus, for ozone, cumulative daily or long-term average exposures are largely determined by exposures occurring outdoors in the afternoon. The studied effects of exposure misclassification are in the direction of an underestimation of ozone exposure effects and may conceal real effects.

Determining the relationship between ambient levels and personal exposure (Fig. 5.1) is critical in epidemiological studies on the health effects of ozone. The indoor : outdoor ratio is not constant, being affected by the housing and cultural conditions of a given population. Such behaviour may be one explanation for the clearer determination of the effects of ozone during warmer weather, when infiltration is greater, people spend more time outdoors, and ambient measurements consequently reflect personal exposure more precisely.

Fig. 5.1. Exposures driven by ambient concentrations are modified by time-activity and infiltration



Exposure versus ambient measurements

The spatial variability of ozone levels may be low within large areas. However, there are gradients within cities, owing to the reaction of ozone with NO_x emitted from traffic and other combustion sources. There may even be a substantial variation between neighbouring residential areas, as measured by front-door samples (134). In addition, there is a strong diurnal variation, with the highest levels usually in the afternoon. Further, as ozone is a highly reactive gas, it is very quickly removed by reactions with surfaces and other chemicals in the air. The surface-to-volume ratio and the proportion of fleecy surfaces such as upholstered furniture, carpets and curtains affect ozone removal. The indoor : outdoor ratio for ozone is usually in the range 0.2–0.7. It may be lower in a closed bedroom and higher if windows are kept open. Owing to the small-scale spatial variation and indoor–outdoor differences, short-term personal exposure measurements are not well-correlated with ambient fixed-site measurements (134). The use of outdoor ozone concentrations from fixed-site monitors as a measure of short-term ozone exposure in epidemiological studies may, therefore, result in misclassification, both in studies with temporal and in those with spatial contrasts.

In one study, the temporal correlation was found to vary among subjects according to the activity pattern, geographical variables, home variables such as ventilation, and distance from the monitoring station and traffic (135). Most people spend 90% or more of their time indoors, where ozone levels are lower than those outdoors. Thus the exposure dose would be considerably lower when more time is spent inside. This is supported by epidemiological observations showing that the effect of ambient concentrations is weaker in cities with a higher usage of air conditioning (136).

In spite of the poor temporal correlation on the individual level, in the largest follow-up study on ozone exposure, the differences in average levels between communities were similar when outdoor measurements and personal measurements were used, though only during the warm season. This is probably due to the fact that people spend more time outdoors in the summer and that the differences between outdoor and indoor levels are smaller because windows are left open. This finding is relevant for studies on long-term effects because in summer

the outdoor measurement provides a valid estimate of spatial variation, provided time spent by people in the different areas is measured (137). It has also been shown (65,137,138) that using air conditioning decreases the personal ozone exposure level and also its correlation with outdoor measurements. Most of these random misclassification effects cause true effects to be interpreted as less strong (139). It is, however, possible that the exposure errors are correlated to the exposure level, which would lead to a positive or negative bias. Systematic errors may also occur in studies of urban areas where the ozone levels are substantially lower in the city centres (spatial error). A few epidemiological studies have explicitly assessed the consequences of the poor correlation between personal exposure and the commonly used ozone levels measured at fixed sites. The misclassification error was found to bias the effect estimates towards the null hypothesis (140,141).

Some of the studies on long-term effects have tried to reduce spatial or temporal error by adding additional information to the outdoor measurements. In the AHSMOG study, individual cumulative exposure was calculated using monthly measurements from air monitoring stations in California and distance from residence and work to the stations. This interpolation method was found to increase the validity of the exposure estimates (142). One Austrian study also calculated an individual ozone concentration by weighting the outdoor measurements by the time spent in the area (143).

To account for the difference between ambient and personal exposure, other studies used additional information (such as activity patterns) to improve personal exposure estimates based on fixed-site measurements. The studied effects of exposure misclassification are in the direction of underestimating ozone exposure effects and may conceal real effects. However, even if misclassification problems are likely, there is no easy way to adjust exposure–response functions obtained in time-series studies of daily number of hospital admissions or deaths.

Exposures in rural vs urban areas

A large and growing proportion of the European population live in cities, which is why a health impact assessment needs to estimate ozone exposure in urban areas in a way that corresponds to the fixed-site measurements that have been used in epidemiological studies. Studies of short-term effects of ozone on hospital admissions and mortality are, for reasons of efficiency, mainly conducted in larger urban areas with sufficient daily cases, while panel studies may be conducted in more rural settings. To match most of the evidentiary health studies, it is regarded as sufficient to use one average ozone concentration level per city.

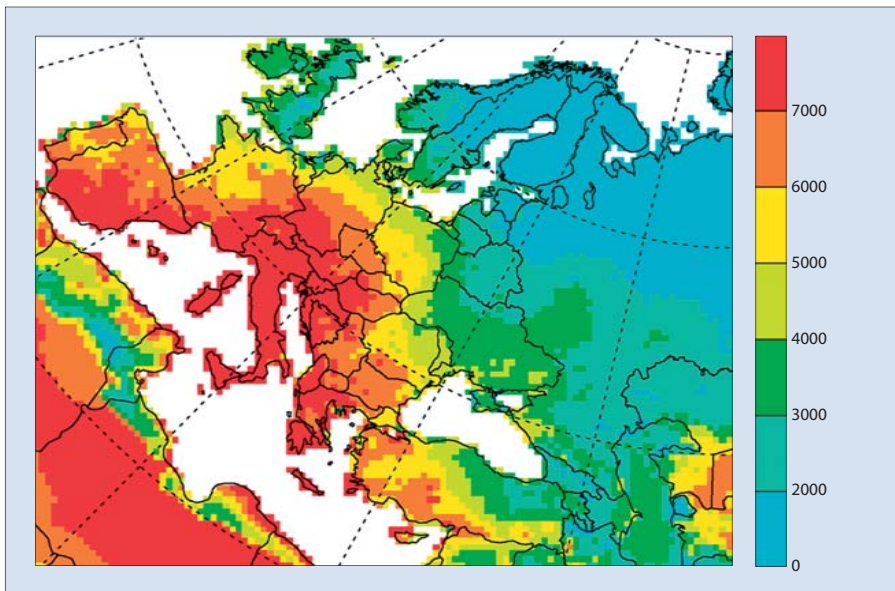
Several factors determine the levels of ozone found at a certain location, as described in Chapter 4. They range from global and regional influences to local conditions, including presence of NO_x sources.

Close to the source, emission of NO_x will result in a temporary reduction of ozone through titration (144). Thus, ozone observations from traffic-oriented measurement stations may largely underestimate the average urban background level and population exposure. A background station in the downwind plumes may, on the other hand, report higher levels than the average in the city.

Population exposure modelling

For calculations of SOMO35 relevant to population exposure, the main uncertainty is probably that related to the EMEP grid size (145). Ozone concentrations in urban areas are usually substantially lower than those in rural areas, and we cannot capture this in a model with grid sizes measuring tens of kilometres. In addition, ozone formation is a non-linear process, and finer grid sizes would presumably lead to a more accurate treatment of the ozone chemistry, and hence of responses to emission control. Uncertainties concerning emissions are discussed in Chapter 3. In addition to anthropogenic emissions, a major uncertainty for ozone formation is that of biogenic VOC emissions (146,147). Uncertainties concerning levels arise from the high threshold value (148) and when comparing ozone levels at the top of a vegetation canopy with the typical measurement heights (149). Finally, an important factor in determining levels of SOMO35 and AOT40 in 2010 and beyond is the influence of increasing levels of tropospheric ozone on the results. The assumed increase of $6 \mu\text{g}/\text{m}^3$ over the mean 1990s level seems consistent with the available data. Nevertheless, it should be noted that both measurements and model calculations cover a wide range (82,150,151).

Fig. 5.2. Average SOMO35 values ($\mu\text{g}/\text{m}^3 \cdot \text{days}$) in Europe for 1995–2002



Geographical distribution and trend in SOMO35

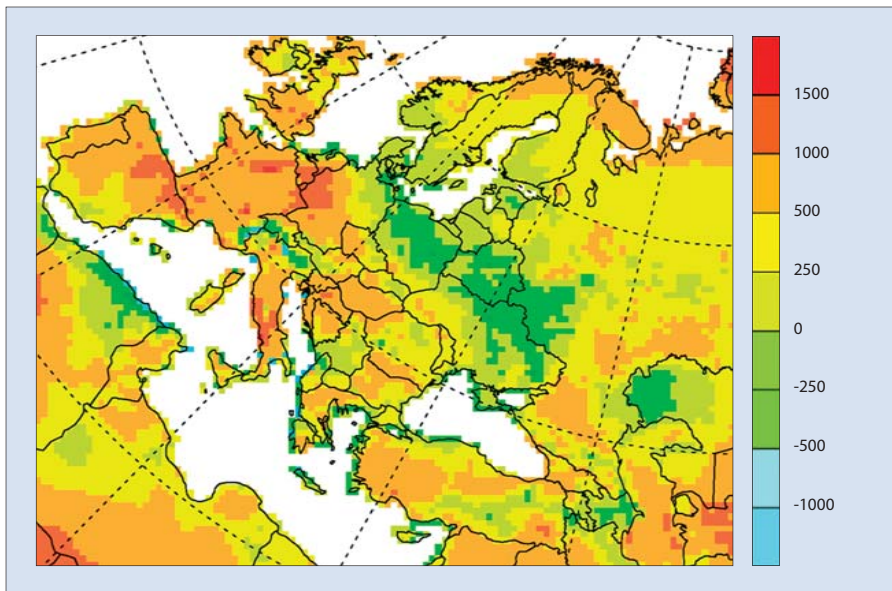
Fig. 5.2 depicts SOMO35 values across Europe as an 8-year average for the period 1995–2002. Maximum values of over 14 000 $\mu\text{g}/\text{m}^3\cdot\text{days}$ are seen over northern Italy. SOMO35 values over much of central and southern Europe exceed 6000 $\mu\text{g}/\text{m}^3\cdot\text{days}$ but decrease further towards northern and eastern Europe. SOMO35 values vary significantly from year to year, and Fig. 5.3 shows the difference between SOMO35 values in 2003 and the 8-year average shown in Fig. 5.2. Clearly, SOMO35 values are generally higher in 2003, especially in France, Italy and Spain. Further examples can be found in Tarrasón et al. (152) and van Loon et al. (153).

Projected exposure trends for 2010–2020

To more systematically evaluate the effects of emissions and boundary conditions for the period 2000–2010, two further tests were conducted using 2000 meteorology but with projected emissions for 2010 (145). The change in background concentration was estimated using two scenarios, one assuming no change and another including a 6- $\mu\text{g}/\text{m}^3$ increase compared to the mean background concentrations employed during the 1990s.

At first sight, the results of both 2010 scenarios look rather similar to that of the 2000 simulations, although lower SOMO35 values are apparent in many areas, for example over northern Italy and Spain. The scenario with increased background levels shows decreases in central Europe, but increases elsewhere (e.g. for

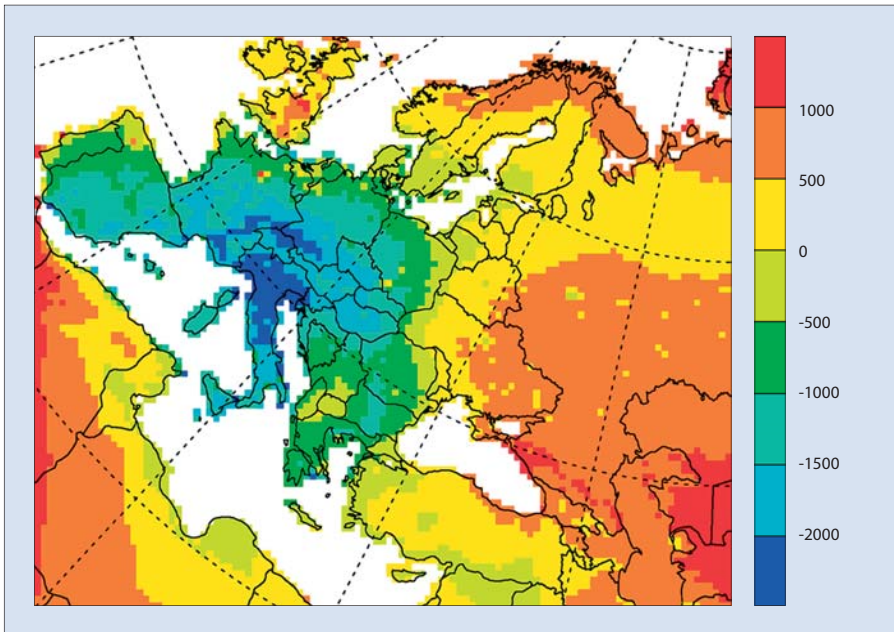
Fig. 5.3. Calculated difference between SOMO35 values ($\mu\text{g}/\text{m}^3\cdot\text{days}$) in 2003 and the 8-year average values presented in Fig. 5.2



the United Kingdom and around the Black Sea). The scenario using 2000 background levels shows greater reductions in SOMO35 over a larger area of Europe. Only a few regions show notable increases in SOMO35, explained by a reduction in the NO_x titration effect over the United Kingdom (as NO_x emissions are reduced) and by increased NO_x emissions in many parts of southeastern Europe.

Finally, Fig. 5.4 illustrates the expected changes in SOMO35 for 2020 compared to similar calculations with 2000 emissions. In some areas, such as parts of the Russian Federation and Scandinavia, SOMO35 levels are seen to increase in 2020 compared to 2000, although absolute levels are relatively low (around $2000 \mu\text{g}/\text{m}^3\cdot\text{days}$) in both cases. In those areas that had high levels of SOMO35 in 2000 (e.g. Italy and indeed much of southern Europe), 2020 levels are seen to be significantly lower than 2000 levels, although this still leaves levels of around $4000\text{--}5000 \mu\text{g}/\text{m}^3\cdot\text{days}$ in these areas. Further details of these calculations are presented in Jonson et al. (154).

Fig. 5.4. Calculated changes in SOMO35 values ($\mu\text{g}/\text{m}^3\cdot\text{days}$) by 2020 compared to 2000 emissions (meteorology from 1997)



6. Risk estimates

KEY MESSAGES

- Current exposures to ozone in Europe are associated with premature mortality and morbidity. Effects include 21 000 premature deaths, 14 000 hospital admissions for respiratory disease and more than 100 million person-days of restricted activity per year in the EU 25. These figures are underestimates, as they do not account for possible effects at levels below $70 \mu\text{g}/\text{m}^3$.
- Reduction in ozone exposure resulting from current policies, and thus in the health impact by 2020, is estimated to be small.
- Population ageing will increase susceptible groups and background risks in Europe in the foreseeable future.
- Current estimates consider acute effects only. Owing to a lack of evidence, the possibility of chronic effects and reductions in life expectancy cannot be ruled out.

This chapter provides an overview of the approach selected to quantify the health effects of exposure to ozone in Europe. The main steps in conducting a health impact assessment are outlined in Fig. 6.1, which also sets out the roles of air quality monitoring and modelling in different phases of the process. Monitoring data are needed for reliable estimation of the concentration–response function (relative risk). When the relative risk has been estimated, monitoring and modelling can be used to estimate exposures and associated health effects. Only modelling, however, can be used in defining policies for reducing health effects attributable to air pollution. While health impact assessment needs to be based on the exposure proxy, typically pollutant levels in the ambient air at central monitoring

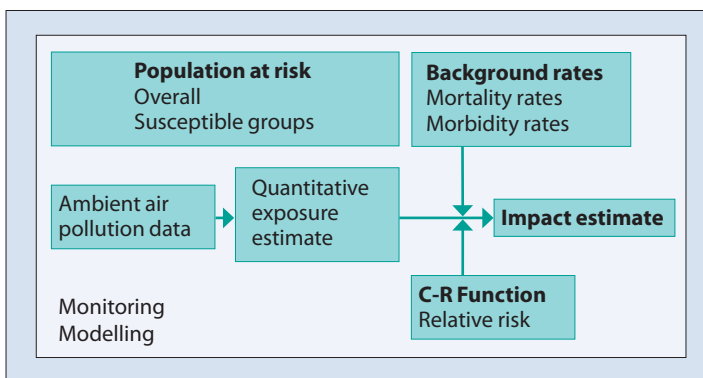


Fig. 6.1. The steps in a health impact assessment

sites, the policy optimization process also needs to consider how policies affect population exposure other than by affecting ambient air concentrations (such as by changing the spatial distribution of the population in areas affected by the policy).

The sources of information for deriving air pollution data and exposure estimates are described in previous chapters. The selection of appropriate health end-points for which impact estimates can be derived, air pollution indicators and concentration–response functions are outlined in this chapter. The methodology was also reviewed, discussed and approved by experts and stakeholders at two meetings of the Joint WHO/Convention Task Force on the Health Aspects of Air Pollution (79,155).

Effects of ozone on mortality

Mortality as the main end-point

The most important types of health effect in the scientific literature that are associated with ozone exposure are discussed in Chapter 2. Besides premature death, these effects include increases in a number of types of morbidity (Table 6.1).

Table 6.1. Health effects associated with ozone exposure

Short-term exposure	Long-term exposure
Adverse effects on pulmonary function	Reduction in lung function growth
Lung inflammatory reactions	
Adverse effects on respiratory symptoms	
Increased medication usage	
Increase in hospital admissions	
Increase in death rates	

There are several reasons for asserting that mortality is the most robust, reliable and significant indicator of the overall health effects of ozone air pollution. The main factors making premature death the first choice of outcome in this respect are discussed in detail by WHO (3,33) and are summarized below.

1. *The evidence is strong.* There is a large a number of (European and non-European) time-series studies showing an association between mortality and short-term exposure to ozone, i.e. daily variations in ozone.
2. *There is a consistent definition of the end-point.* Death is a well-defined event that is registered in all European countries. For this reason, epidemiologists have frequently assessed the effect of air pollution on mortality (either all-cause or cause-specific). Other outcomes, such as bronchitis, are subject to very large variations in severity, and without such qualification their health impact is difficult to assess. The definitions of other possible health outcomes,

such as restricted activity days or use of primary care services, are likely to vary with national culture and among health care systems.

3. *Baseline occurrence rates are available.* Mortality data are collected or estimated using consistent methods, for all European countries, and the data can be obtained relatively easily. This is generally not the case for most important morbidity outcomes, such as number of asthma attacks or hospital admissions.
4. *The end-point is important in terms of health impact.* Air pollution has an impact on health end-points other than mortality; nevertheless, there are studies showing that mortality, quantified as numbers of deaths or reduction in life expectancy, is the most important health impact in terms both of disability-adjusted life-years (DALYs) (156) and the economic impact (157).

It should be stressed that the focus on mortality does not imply that there are no other relevant health end-points, some of them posing a considerable health burden. These are also considered in this chapter.

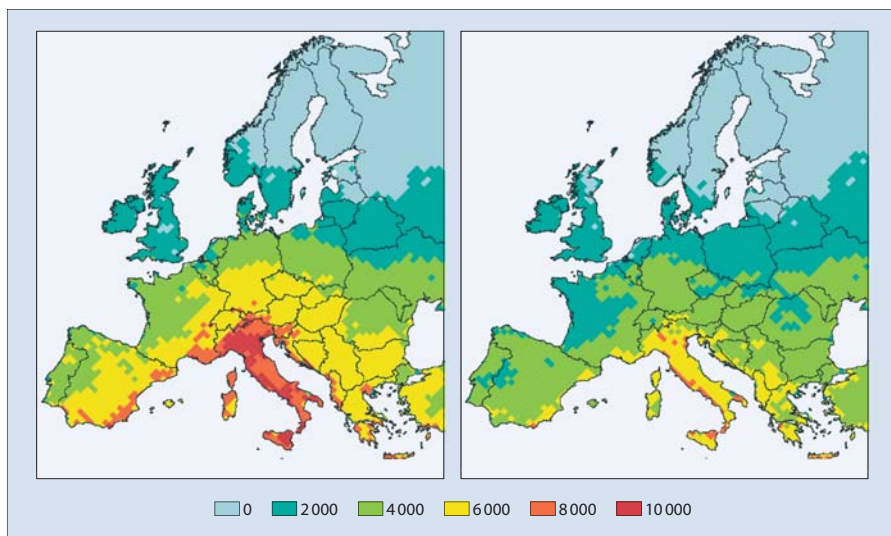
Estimates of premature mortality

It has been known for several years that exposure to ground-level ozone impairs health, and a range of morbidity end-points have been associated with increased exposure. The WHO systematic review of health aspects of air quality in Europe (158) as well as the recent update of WHO's air quality guidelines (2) confirmed the health relevance of exposure to ozone. The review found that recent epidemiological studies have strengthened the evidence that effects of ozone observed in short-term studies on pulmonary function, lung inflammation, respiratory symptoms, morbidity and mortality are independent of those of other pollutants, particularly in the summer.

The joint WHO/Convention Task Force, at its seventh meeting in May 2004 (79), developed specific recommendations concerning the inclusion of ozone-related mortality into the RAINS modelling framework. The key points of these recommendations are as follows.

- Owing to the lack of consistent baseline morbidity data, a Europe-wide assessment of the health effects of ozone should focus on mortality.
- This assessment should assume a linear relative risk (concentration–response function) of 1.003 for a 10- $\mu\text{g}/\text{m}^3$ increase in the maximum daily 8-hour average (95% CI 1.001–1.004) derived from a commissioned WHO meta-analysis (21).
- Effects of ozone on mortality are calculated from the maximum daily 8-hour average.
- Although current evidence was insufficient to derive a level below which ozone has no effect on mortality, as a conservative assumption a cut-off at 70 $\mu\text{g}/\text{m}^3$ (maximum daily 8-hour average) was recommended for the analysis.

Fig. 6.2. Rural ozone concentrations expressed as SOMO35 for 2000 (left) and 2020 (right) modelled as averages of four runs with meteorological data for 1997, 1999, 2000 and 2003



Source: Amann et al. (67).

The RAINS analysis, and the later associated analyses of the CAFE Cost–Benefit Analysis (CBA) project, both apply the SOMO35-based methodology (see Box 4.2 and page 56) to quantify the changes in premature mortality that are attributable to the projected reductions in ozone precursor emissions (NO_x and VOCs). The Eulerian EMEP model was used to calculate the SOMO35 exposure indicator for the CAFE programme emission projections, both for the beginning of the current decade and for 2020 (Fig. 6.2).

Using the methodology outlined above, the changes in premature mortality that are attributable to the projected reductions in ozone precursor emissions have been estimated for EU25. For health impacts attributable to ozone, RAINS calculates the number of premature deaths as SOMO35 on a grid basis and sums them up to a country balance. Health impacts were not tabulated for other eastern European countries (i.e. Belarus and Ukraine) or the European part of the Russian Federation. The later CAFE CBA analyses used a similar methodology. Results reported here are from the CAFE CBA work, and in particular from Watkiss et al. (159).

Overall, for average meteorological conditions, the expected decline in ground-level ozone under CLE, and taking account of climate change, is estimated to reduce premature mortality between 2000 and 2020 by only some 600 cases per year compared to approximately 21 400 cases computed for 2000 (Table 6.2). Markedly larger (around 40%) reductions could be achieved by implementing the MTRF scenario. Corresponding data by country are given in Fig. 6.3 and Table 6.2.

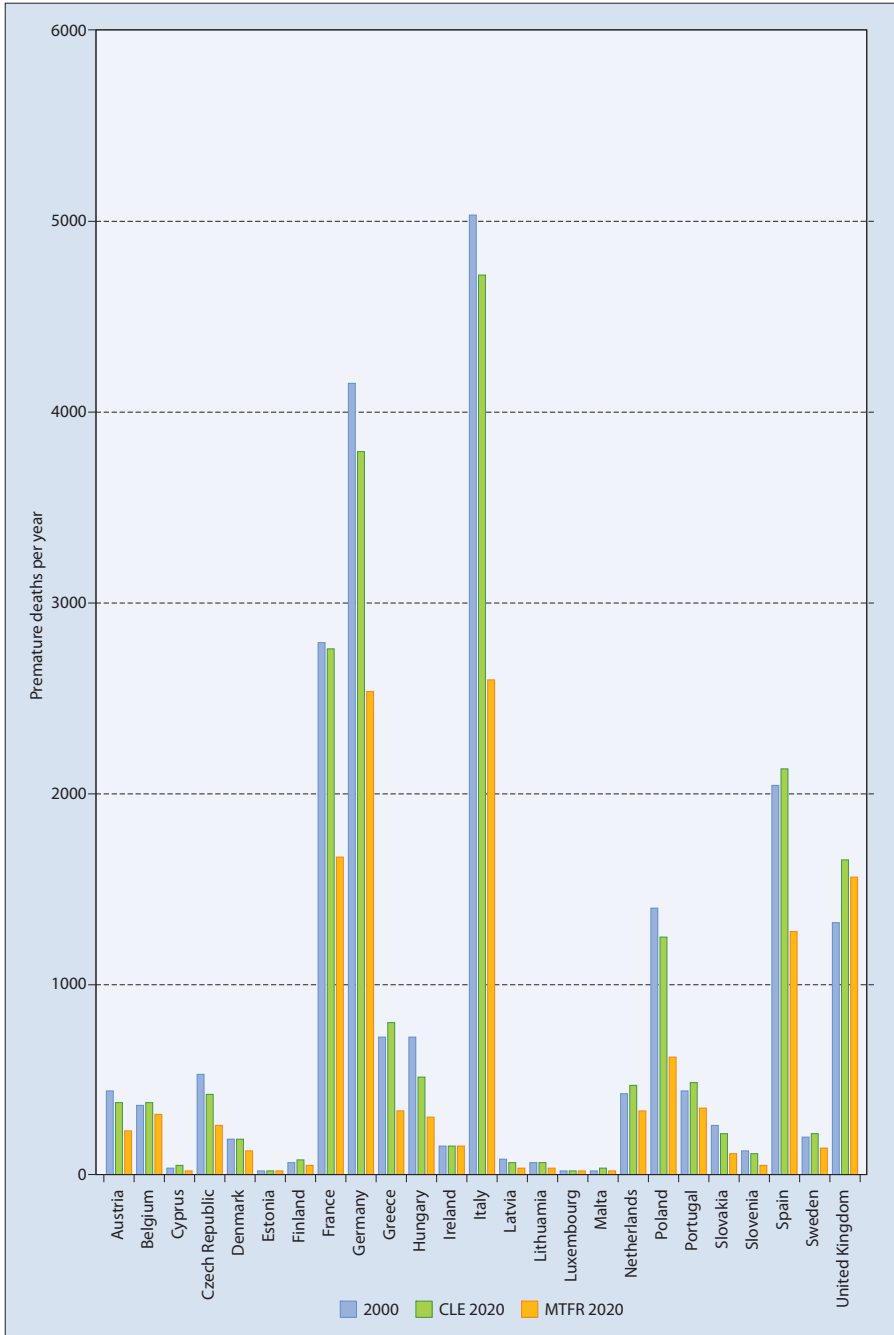
Table 6.2. Estimates of cases of premature death per year (to three significant digits) attributable to ozone, based on regional-scale ozone calculations (50 × 50 km) and applying the meteorological conditions of 1997

Country	2000	2020 (CLE scenario)	2020 (MTFR scenario)
Austria	435	369	220
Belgium	364	381	309
Cyprus	33	42	19
Czech Republic	521	414	257
Denmark	178	175	126
Estonia	22	20	13
Finland	58	71	39
France	2 780	2 750	1 655
Germany	4 150	3 790	2 535
Greece	711	789	334
Hungary	720	515	300
Ireland	71	96	68
Italy	5 030	4 710	2 583
Latvia	74	67	35
Lithuania	55	53	29
Luxembourg	16	16	20
Malta	21	25	15
Netherlands	415	460	336
Poland	1 390	1 240	609
Portugal	439	485	350
Slovakia	248	209	99
Slovenia	119	105	52
Spain	2 030	2 120	1 271
Sweden	196	206	135
United Kingdom	1 320	1 650	1 554
Total EU25	21 400	20 800	12 962

Source: Watkiss et al. (159).

These estimates should be interpreted with caution, however, because of various uncertainties (which to some extent could be reduced by further analysis) and because of limitations in the information on mortality that can be obtained from the underlying time-series studies assessing the effects of changes in ozone levels on daily mortality rates. By their nature, such studies show how the numbers of daily deaths vary in relation to daily ozone concentrations on the same or immediately preceding days, but they cannot provide any direct information on how much the deaths have been brought forward, i.e. by how much lives have been shortened as a result of exposure to ground-level ozone. Mechanistic reasoning suggests that, in general, these are deaths of people with already serious

Fig. 6.3. Estimates of cases of premature death per year attributable to ozone for the CAFE baseline scenario (2000) and predictions based on CLE and MTRF scenarios with climate change effects included



Note: These calculations are based on regional-scale ozone calculations (50 × 50 km) and averaged over the meteorological conditions of four years (1997, 1999, 2000, 2003).

Source: Watkiss et al. (159).

(though possibly undiagnosed) disease. This implies that the lives of some of these people are shortened by at most a few days while others, had they survived, might have lived apparently healthy lives for several years. At present, it is not possible to quantify reliably the importance of the associated life shortening for these premature deaths. To estimate the number of years of life lost due to exposure, ExternE (160) and Rabl (161) assumed that life is shortened by an average of six months as a result of exposure. However, following comments from the reviewers, CAFE CBA followed Levy et al. (162) in assuming that the arithmetic mean of life-shortening is one year and that the distribution of life-shortening may be heavily skewed, i.e. has lower median value (163).

Furthermore, the influence of the selected cut-off value ($70 \mu\text{g}/\text{m}^3$) on the outcome needs to be further explored in the future. The exposure indicator (SOMO35) is based on applying a conservative approach to integrated assessment modelling: because of the uncertainties in the shape of the concentration–response function at very low ozone concentrations, it ignores any effects at these low concentrations. Preliminary results suggest that using a lower cut-off value, or none, has a major effect on absolute numbers of ozone-related premature deaths but that the effect on the change from 2000 to 2020 is not very large.

Estimation of impacts on morbidity

As noted in AIRNET (164) and CAFE CBA (163), there are two different traditions in quantifying the effects of air pollution on morbidity. These reflect different purposes in carrying out the analysis and different uses of the results. One approach, such as that used by COMEAP (165) and APHEIS (166), quantifies only those end-points where there are strongly reliable data both for concentration–response functions and for background rates of morbidity. This is useful in showing that there is a public health problem of at least the magnitude quantified; it can help provide general (i.e. not policy-specific) motivation to protect health by reducing air pollution. Its main drawback is that, by including only those effects for which there is a strong evidence base for quantification, it systematically underestimates the overall effects of air pollution on health. When used in cost-benefit analyses, it is anti-precautionary.

The second approach (157,160,167,168) aims to quantify all health end-points where, on balance of probability, the relevant air pollutant has an effect. This implies quantification of a wider range of end-points and, for some of the impacts included in the second quantification, a greater uncertainty in the concentration–response function and/or in background rates than would be acceptable under the first, more restrictive, approach. Nevertheless, the approach as a whole gives a fairer and more realistic assessment of the overall effects of air pollution. It is the appropriate strategy when comparing the costs and benefits of specific policies or developments that affect air pollution, and so Hurley et al. (163) adopted it for CAFE CBA. Also, they focused on studies of incidence rather than preva-

lence, so that the benefits of reducing pollution could more easily be expressed as annual benefits, for comparison with annual costs. Moreover, they did not include lung function end-points because there are no suitable studies linking them with monetary valuation. Hence they did not quantify any effects on morbidity of long-term exposure to ground-level ozone (see Table 6.1).

Implementation, including impact functions

The two health impact assessment approaches use the same technical methods in estimating the effects of ambient ozone on any particular morbidity end-point, methods that are very similar to those used in estimating attributable deaths. As described earlier, these methods use a concentration–response function expressed as percentage change in end-point per $(10) \mu\text{g}/\text{m}^3$ (ozone) and link this with (a) the background rates of the health end-point in the target population, expressed as new cases (or events) per year per unit population (say per 100 000 people); (b) the population size; and (c) the relevant pollution increment expressed as $\mu\text{g}/\text{m}^3$ ozone. Results are then expressed as estimated new or “extra” cases, events or days per year attributed to ozone. As was done earlier for mortality, the core analyses of CAFE CBA quantified adverse health effects of ozone only at ozone concentrations exceeding a cut-off of $70 \mu\text{g}/\text{m}^3$ maximum daily 8-hour average, irrespective of season, and thus use SOMO35 ozone estimates.

When the concentration–response function is derived from Poisson regression or related analyses, such as time-series studies of hospital admissions, it is easy and natural to express the relative risk as the percentage change in background rates of incidence (or prevalence). When the concentration–response function is derived from logistic regression analyses, such as in panel studies of daily occurrence of symptoms or individuals’ use of medication, then the estimated relative risk applies to the odds of occurrence, when what is needed is the relative change in probability. When the occurrence is rare, with probability of (say) less than one in ten, odds and probability are interchangeable with little error. A percentage change in probability can also be derived for more frequently occurring events, but its value depends on the background rates used. For some examples, see Hurley et al. (163).

The percentage change in probability can be combined with background rates to give a single impact function expressed as:

**number of (new) cases, events or days per unit population (say per 100 000 people)
per $(10) \mu\text{g}/\text{m}^3$ ozone per annum.**

This impact function can then be linked, as before, with population size and the relevant pollution increment to give the estimated number of annual occurrences in the target population. This is convenient in implementation.

Dealing with missing data on background rates

For many health end-points, reliable data on background rates of morbidity in the EU25 target population are not readily available, because they are not collected routinely or, if collected, may be to different protocols and standards in different locations or not readily accessible. One strategy then is to use other general epidemiological studies of that health end-point – not necessarily studies on air pollution and health – to provide estimates of background rates. Examples of such studies are the International Study of Asthma and Allergies in Children (ISAAC) (169) and, for adults, the European Community Respiratory Health Study (ECRHS) (170).

Another approach is to estimate an impact function from where the relevant epidemiological studies were carried out and then transfer and use it for quantification in the wider European target population. Clearly, the reliability of that quantification depends on how transferable the impact function is, that is, how stable it is spatially. The approach is, however, well-established in health impact assessment practice (157,160,167). The two approaches were used (for different end-points) for CAFE CBA; otherwise, few if any morbidity end-points would have been quantifiable.

Concentration–response functions for morbidity

Respiratory hospital admissions (RHAs; ICD-9 460–519)

Anderson et al. (21), in their meta-analyses for WHO and CAFE, used results from five cities in western Europe to estimate the change in all RHAs in various age groups in relation to daily variations in ozone (maximum daily 8-hour average). The result of the meta-analysis for elderly people was close to statistical significance, giving a concentration–response function of 0.5% (95% CI –0.2–1.2) per 10 $\mu\text{g}/\text{m}^3$ ozone (maximum daily 8-hour average) in people aged ≥ 65 years. Background rates were taken from the APHEIS second year report (171), where rates for emergency hospital admissions from eight cities in western and northern Europe gave an average incidence (unweighted arithmetic mean) of 2496 per 100 000. Together, the concentration–response function and incidence imply an impact function:

annual rate of attributable emergency RHAs
per 100 000 people aged ≥ 65 years = 12.5 (95% CI –5.0–30.0) per 10 $\mu\text{g}/\text{m}^3$ ozone
(maximum daily 8-hour average).

The effect of quantifiable uncertainties in background rates, concentration–response function and monetary valuation was explored quantitatively, using Monte Carlo methods, by Holland et al. (172).

The main unquantifiable uncertainty is the extent to which the extra hospital admissions in days following higher air pollution episodes are genuinely addi-

tional admissions, rather than the bringing forward in time of admissions that might soon have occurred in any case (165).

Minor restricted activity days

Ostro & Rothschild (173) used data on adults aged 18–64 years from six consecutive years (1976–1981) of the United States Health Interview Study (HIS), a multi-stage probability sample of 50 000 households from metropolitan areas throughout the country. Within the HIS, minor restricted activity days (RADs) do not involve loss of work or bed disability, but do include some noticeable limitation on “normal” activity.

For current urban workers, Ostro & Rothschild (173) found relationships between minor RADs (though not respiratory RADs) and ozone (two-week averages of the daily one-hour maximum, in $\mu\text{g}/\text{m}^3$). The weighted mean coefficient for ozone, adjusted for $\text{PM}_{2.5}$, from separate analyses of each of the six years 1976–1981 (weights inversely proportional to coefficient variances) was derived as 0.00111 (SE 0.00034), giving an increase of:

1.48% (95% CI 0.57–2.38) per 10 $\mu\text{g}/\text{m}^3$ ozone (8-hour daily average).

Ostro & Rothschild (173) reported a mean background rate of 7.8 minor RADs per year among employed people aged 18–64 years. Concentration–response function and background rate were linked to give an estimated impact function:

increase in minor RADs = 115 (95% CI 44–186) per 10 $\mu\text{g}/\text{m}^3$ ozone (8-hour daily average) per 1000 adults aged 18–64 per year.

Medication (bronchodilator) usage by people with asthma

WHO (33) concluded that there is sufficient evidence to assume a causal relationship between air pollution exposure and aggravation of asthma in children. One way that such a relationship may show is via increased medication usage.

However, the WHO meta-analysis (21) identified only one study in Europe linking daily ozone (8-hour daily average) and medication use in children. This study, of 82 children with medically diagnosed asthma in Paris in early summer 1996, found an odds ratio of 1.41 (95% CI 1.05–1.89) per 10 $\mu\text{g}/\text{m}^3$ ozone for increased (supplementary) use of bronchodilators when analyses were restricted to days on which no corticosteroids were used by the children (174). The study may well be unrepresentative: the odds ratio is very high compared with other end-points, and it may be best to consider it as an upper limit. The end-point was included in CAFE CBA for completeness, given WHO’s overall assessment of the relationship.

Hurley et al. (163) use results from Gielen et al. (175), from the ISAAC study (169) and from Just et al. (174) to estimate (a) that the mean daily prevalence of

bronchodilator usage in qualifying children (all of whom were taking daily anti-asthma treatment) is about 40% and (b) the number of qualifying days annually per 1000 children in the general population. This led to an impact function as follows:

annual change in days of bronchodilator usage per 10 $\mu\text{g}/\text{m}^3$ ozone per 1000 children aged 5–14 years (general population) = 124 (95% CI 18–227) in northern and eastern Europe and 310 (95% CI 44–569) in western Europe.

The different functions reflect the higher prevalence of childhood asthma in western than in northern and eastern Europe (169).

For adults, the WHO meta-analysis (21) identified two relevant studies, only one of which (169) gave results in a relevant metric (daily maximum 8-hour moving average) of ozone. The relationship of ozone to daily prevalence of bronchodilator usage was positive (odds ratio 1.009 per 10 $\mu\text{g}/\text{m}^3$ ozone) but not statistically significant (95% CI 0.997–1.020) at the selected lag of one day. However, when seven-day cumulative ozone was considered, the estimated effect was higher and statistically significant. Hiltermann et al. (176) concluded that bronchodilator use was associated with ozone.

The summertime mean daily prevalence of bronchodilator use by people with asthma was taken as 32%. Data from the ECRHS were used to estimate that, across Europe, about 4.5% of adults have asthma of a severity comparable to that of the Dutch panels on which the concentration–response function was based (170). These data were linked to give an estimated impact function:

change in days of bronchodilator use of 730 (95% CI –255–1570) per 10 $\mu\text{g}/\text{m}^3$ ozone per 1000 adults aged ≥ 20 years with well-established asthma (say 4.5% of the adult population).

Acute respiratory symptoms

Most available studies are based on panels followed up during the spring or summer months when there is a greater possibility of detecting an adverse effect of ozone, because ozone levels are higher and daily variations in ozone are greater than in winter. Also, children spend more time outdoors during this period.

For children, the WHO meta-analysis identified only one study linking daily variations in ozone with cough in asthmatic children (174). The relationship was positive but not statistically significant. However, the effects of ozone on health are currently being reviewed by COMEAP in the United Kingdom and among their working conclusions (H. Walton, unpublished data, 2008) are the following.

- There is convincing evidence that daily variations in ozone are associated with lower respiratory symptoms (LRS), including cough.

- Evidence for an association with upper respiratory symptoms is more equivocal.
- Effects on LRS, cough or phlegm production are not restricted to people with chronic respiratory symptoms such as asthma. Indeed, there is no strong evidence that relative risks or odds ratios of daily symptoms linked with ozone are higher among people with chronic respiratory disease than among the general population.

On that basis, CAFE CBA used results from a small general population study in the period April–June of 91 children in Armentières, northern France, in relation to ozone and other pollutants (177). In separate analyses, daily prevalence of cough and phlegm and of breathing problems (difficulty in breathing, wheeze and asthma, i.e. LRS, excluding cough) were associated (though not statistically significantly) with maximum daily 8-hour ozone. The relevant concentration–response functions were:

- for cough: odds ratio 1.05 (95% CI 0.99–1.12) per 10 $\mu\text{g}/\text{m}^3$ ozone;
- for LRS, excluding cough: odds ratio 1.03 (95% CI 0.92–1.15) per 10 $\mu\text{g}/\text{m}^3$ ozone.

Hoek & Brunekreef (178) reported mean daily prevalences of 5.4% for cough and 1.5% for LRS (defined as wheeze, chest tightness, shortness of breath or phlegm production) in a general population sample of 300 children aged 7–11 years in the Netherlands, studied between late March and the end of June. These data were linked to give the following impact functions:

a change of 0.93 (95% CI –0.19–2.22) cough days and 0.16 (95% CI –0.43–0.81) days of LRS (excluding cough) per child aged 5–14 years per 10 $\mu\text{g}/\text{m}^3$ ozone per year.

For adults, the WHO meta-analysis (21) identified only two relevant studies when reviewing ozone in relation to daily cough in people with chronic respiratory disease. Neither showed a statistically significant positive association (176, 179).

Issues of uncertainty are addressed, as for other end-points, in CAFE CBA (172). The main uncertainty issues are as follows.

- The estimates are based on the HIS study only, albeit that this is a large-scale countrywide study with separate analyses of each of six years of data.
- There may be transferability problems from the United States to Europe, because minor RADs are culture-related variables. However, background rates for RADs used in the United States are similar to those from similar surveys in Canada and lower than those in the United Kingdom.
- It is curious that, for ozone, the relationships are with minor RADs rather than respiratory RADs. However, the number of respiratory RADs will have been lower than that of minor RADs.

- On the other hand, the CAFE CBA estimates may be too low because:
 - the background rates used were for workers, and these may underestimate overall rates in the 18–64-year age group because employed people are on average healthier and better off socioeconomically than those who are unemployed; and
 - in the main CAFE CBA analyses, this impact function was applied to people aged 15–64 years (as in the original study) and it is possible that, had older people been studied, similar results would have been found.

Health effects and cost–benefit analysis

Estimates of the impacts of ozone in 2000 and 2020 were calculated for EU25. The difference between these years was used to estimate the health benefits of the policies to be implemented (159). The estimates for 2020 are based on projections forward in time of two factors:

- ozone concentrations, specifically SOMO35, estimated on the basis that all the emission control legislation that has already been passed will be implemented fully in all EU25 countries;⁴ and
- population size, based on United Nations population projections for various age groups.

The main results are summarized in Table 6.3 (159). Evidence of some benefit (reduced health effects) is shown for all health end-points other than RHAs, where

Table 6.3. Estimates of health effects caused by ozone in 2000 and 2020 and changes between the two years (in 1000s) due to implementation of current emission policies

Health end-point	End-point output	2000 (baseline)	2020 (CLE scenario)	Change
Mortality (all ages)	Premature death (cases)	21	20	–0.60
RHAs among older people (≥65 years)	Cases	14	20	6.1
Minor RADs among adults (15–64 years)	Person-days	54 900	42 400	–11 500
Respiratory medication use in children (0–14 years)	Person-days	21 000	13 000	–8 000
Respiratory medication use in adults (>20 years)	Person-days	8 800	8 200	–660
Cough and LRS in children (0–14 years)	Person-days	108 000	65 000	–43 000

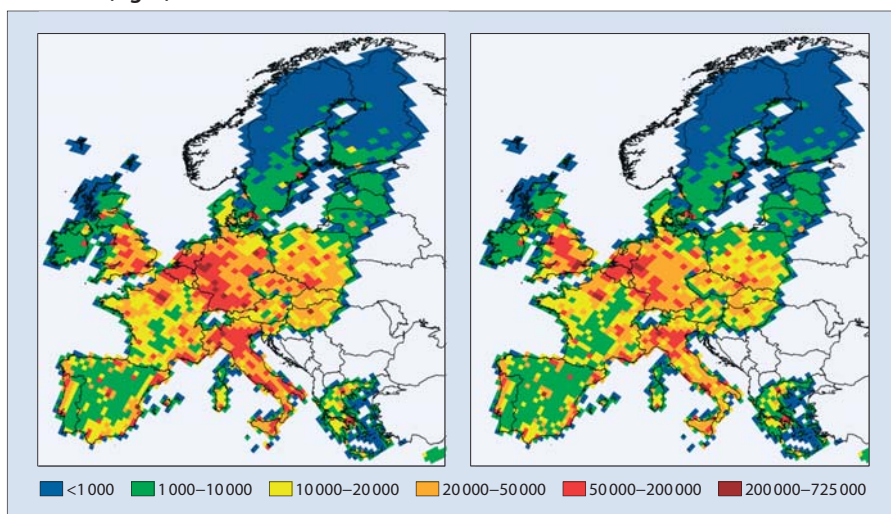
Note: The results are presented to two significant digits.
Source: Watkiss et al. (159).

⁴ This includes legislation dealing with climate policy (policy obligations under the Kyoto Protocol: continued implementation of greenhouse gas reduction policies through to 2020).

the 2020 figure is higher than that of the 2000 baseline. The reason is straightforward: RHAs are the only end-point where effects are estimated only in those aged ≥ 65 years, and this population will be larger in 2020 than in 2000. This gain in the population at risk more than offsets the small reductions in ozone (SOMO35). The largest relative gains occur where impacts are estimated only for younger people or adults age 15–64 years.

The estimates presented in Table 6.3 indicate that ozone is currently responsible for large numbers of days when people experience minor restrictions in personal activity because of health or suffer ozone-related lower respiratory symptoms. By way of illustration, the spatial distribution of the annual ozone-related minor RADs across EU25 in 2000 and 2020 is shown in Fig. 6.4.

Fig. 6.4. Estimated numbers of minor RADs caused by ozone across EU25 in 2000 (left) and 2020 (right)



Source: Watkiss et al. (159).

Transferability between populations

A health impact assessment applies air pollution effect estimates derived from one (evidentiary) population to estimate impacts in another (target) population, based on the assumption that these estimates can be transferred. Care must be taken if one cannot assume that the contribution of various causes of death is similar, if the mixture of pollutants differs, if the baseline health statuses of the populations are not the same, or if exposure ranges do not overlap.

The WHO review on health aspects of air pollution in Europe also assessed the question of possible regional characteristics modifying the effects of air pollution (180). It states: “Potentially this could be a very influential issue since the characteristics of populations, environments and pollution (including particle concentration, size distribution and composition) vary throughout Europe. However,

at this stage there is not sufficient evidence to advocate different guidelines for particles or other priority pollutants in different parts of Europe.”

Evidence on reversibility of the health impacts

Several studies have been published illustrating the benefits, in terms of reduced mortality and morbidity, of large and immediate reductions in ambient PM and/or sulfur dioxide, notably in Dublin (181) and China (Hong Kong SAR) (182). Unfortunately, there have been no similar opportunities to evaluate ozone reduction per se. One reason is that reductions in emissions of ozone precursors (NO_x and VOCs) can result in lower concentrations not only of nitrogen dioxide and ozone but also of fine particles ($\text{PM}_{2.5}$). Without the oxidants generated in the photochemical reaction sequences, there would be a reduction in the oxidation of sulfur dioxide and nitrogen dioxide, which leads to acidic sulfate and fine particles and nitric acid vapour, as well as reduced formation of organic fine particles. It is therefore difficult to assess the benefits to health of reducing ozone alone.

There is nevertheless some evidence of benefit (reversibility of impacts) from two studies. Children in the Southern California cohorts who moved from communities with relatively high PM and ozone concentrations to communities with lower concentrations had better lung function growth than children who remained in those communities (183), while children who moved from communities with relatively low $\text{PM}_{2.5}$ and ozone concentrations to communities with higher concentrations of these pollutants had less lung function growth than those who remained in the cleaner communities. It is not clear, however, whether this is due to changes in ozone or PM. Friedman et al. (184) took advantage of a natural experiment associated with a decrease in ozone exposure in Atlanta, Georgia during the 1996 Olympic Games to demonstrate that acute ozone effects on asthma admissions were substantially reduced. More research is needed in this area, but appropriate opportunities are few.

7. Conclusions

This chapter summarizes the conclusions drawn from evaluation of the material presented in the previous chapters. The conclusions were drawn up during meetings of the Joint WHO/Convention Task Force on the Health Aspects of Air Pollution at which it reviewed consecutive drafts of this report.

Ozone air pollution is a significant health hazard in Europe

Controlled studies in humans indicate that levels of ozone that may be experienced in several areas of the world induce significant functional and biochemical alterations, mostly in the respiratory tract. Although sequential exposures to ozone induce some degree of adaptation, it is plausible that multiple acute injuries may lead to permanent damage to the target organs. Recent epidemiological studies considering larger series or tens of communities, or using other statistical approaches such as case crossover design, have confirmed that ozone is indeed associated to acute adverse health effects, expressed either as morbidity or as mortality.

Animal data and some autopsy studies indicate that chronic exposure to ozone induces significant changes in airways at the level of the terminal and respiratory bronchioli. The reversibility (or not) of such lesions is a point that deserves clarification. Epidemiological evidence of chronic effects is less conclusive, owing mostly to an absence of studies designed specifically to address this question and inherent limitations in characterizing exposure. The studies with the most efficient approaches and more individual assignment of exposure provide new evidence for chronic effects of ozone on small airway function and possibly on asthma.

Ozone was estimated to have caused approximately 21 000 cases of premature death in EU25 in 2000. Moreover, ozone was associated with 14 000 respiratory hospital admissions and affected the daily health of large numbers of people in terms of minor RADs, respiratory medication use (especially in children) and cough and LRS, amounting to between 8 million and 108 million person-days depending on the morbidity outcome in question.

Health effects of long-range transboundary ozone are most likely proportional to the contribution of long-range sources to ozone exposure levels

Since ozone is a gaseous chemical species prevalent in outdoor air and has very few indoor sources, its health effects are expected to be similar regardless of the origin

of precursor emissions. Owing to the time required for photochemical reactions to produce ozone, levels are highest in rural and remote locations. In urban areas, which receive these long-range contributions as background inputs, ozone levels are reduced by other air pollutants, mainly nitrogen oxides. Thus ozone levels are rarely generated locally but have significant regional contributions, being strongly affected by long-range transboundary and intercontinental inputs.

Current policies lead to only a small reduction in risk

Projected estimates of morbidity and mortality for 2020 based on the implementation of current legislation (the CLE scenario) mostly suggest moderate reductions in ozone exposure and associated health effects. Mortality is expected to come down from around 21 000 in 2000 to 20 000 in 2020. Reductions in morbidity are more significant, ranging from approximately 8% (respiratory medication use by adults) to 40% (cough and LRS in children).

Since the elderly are more susceptible to many health impairments, the additional burden associated with air pollution in general, and ozone in particular, translate into larger absolute numbers owing to population ageing. This is highlighted by the estimated >40% increase in respiratory hospital admissions among the elderly.

One of the reasons that the level of ozone and its effects are projected to fall only slightly is the global increase in background ozone levels attributable to LRTAP. Nevertheless, application of the MTRF scenario would result in a significant reduction in the effects of ozone on health.

Key uncertainties, research needs and policy implications

Uncertainty related to the potential impact of ozone on life expectancy is a major limitation of risk assessment, affecting the cost–benefit analysis of actions to reduce ozone exposure. Better knowledge of the magnitude of the risk and of the ozone levels at which long-term exposure may affect mortality and life expectancy will be necessary to reduce this uncertainty. Studies will require more individual assignment of exposure to account for the major discrepancies in indoor and outdoor ozone levels.

There is a wide range of non-lethal health effects attributed to ozone. However, the relevant database is not consolidated and health burden estimates are mostly based on mortality. This may result in a substantial part of the burden being missed, especially in subpopulations with a lower mortality risk, such as children.

Impact estimates depend on the exposure indicator selected and in particular on the cut-off above which the effects are calculated. Better understanding of the shape of the concentration–response function at low levels of exposure and an improved ability of models to estimate low concentrations may affect the decision to use the current cut-off point of 70 $\mu\text{g}/\text{m}^3$.

The shape of the concentration–response function may differ by region, and this may also influence the effect of using a cut-off of $70 \mu\text{g}/\text{m}^3$ (SOMO35) as well as linear concentration–response functions estimated without a cut-off. The results from the APHEA2 study shown in Fig. 2.2 (see page 16) indicate that, for example, the relative increase in daily mortality at $100 \mu\text{g}/\text{m}^3$ may be several times higher than that assumed in current calculations.

The burden of ozone exposure is not evenly distributed in the population, some individuals being more susceptible than others. Deficiencies of the antioxidant defences, asthma, age or high-intensity exercise may modulate the response to exposure. Better characterization of susceptible population groups would allow an improvement in approaches to preventing the impacts and reducing the number of people suffering symptoms of ozone exposure.

There is also a need to continue to assess the well-recognized effects of ozone, such as impacts on daily mortality, so as to better understand significant variations in effects and possibly identify factors modifying the relationship. In particular, the effects of heatwaves related to climate change should be studied, to assess to what extent actions aimed at adapting society to climate change and extreme weather events are effective.

Uncertainty in health impact estimates also depends on the precision of exposure modelling for both current and future situations. In particular, improved methods need to be developed for estimating the exposure of urban populations from model calculations made on a larger scale. Ultimately, estimates of personal exposure would be desirable, both for impact assessment and for use in epidemiological studies. Such improvements will need new developments in the fields of fine-scale emission inventories and fine-scale modelling, as well as observational studies to provide a good basis for the construction and evaluation of estimation methods.

Further uncertainty in projected ozone levels is the contribution from inter-continental ozone transport and the influence of VOCs, NO_x , carbon monoxide and methane emission controls implemented in the northern hemisphere.

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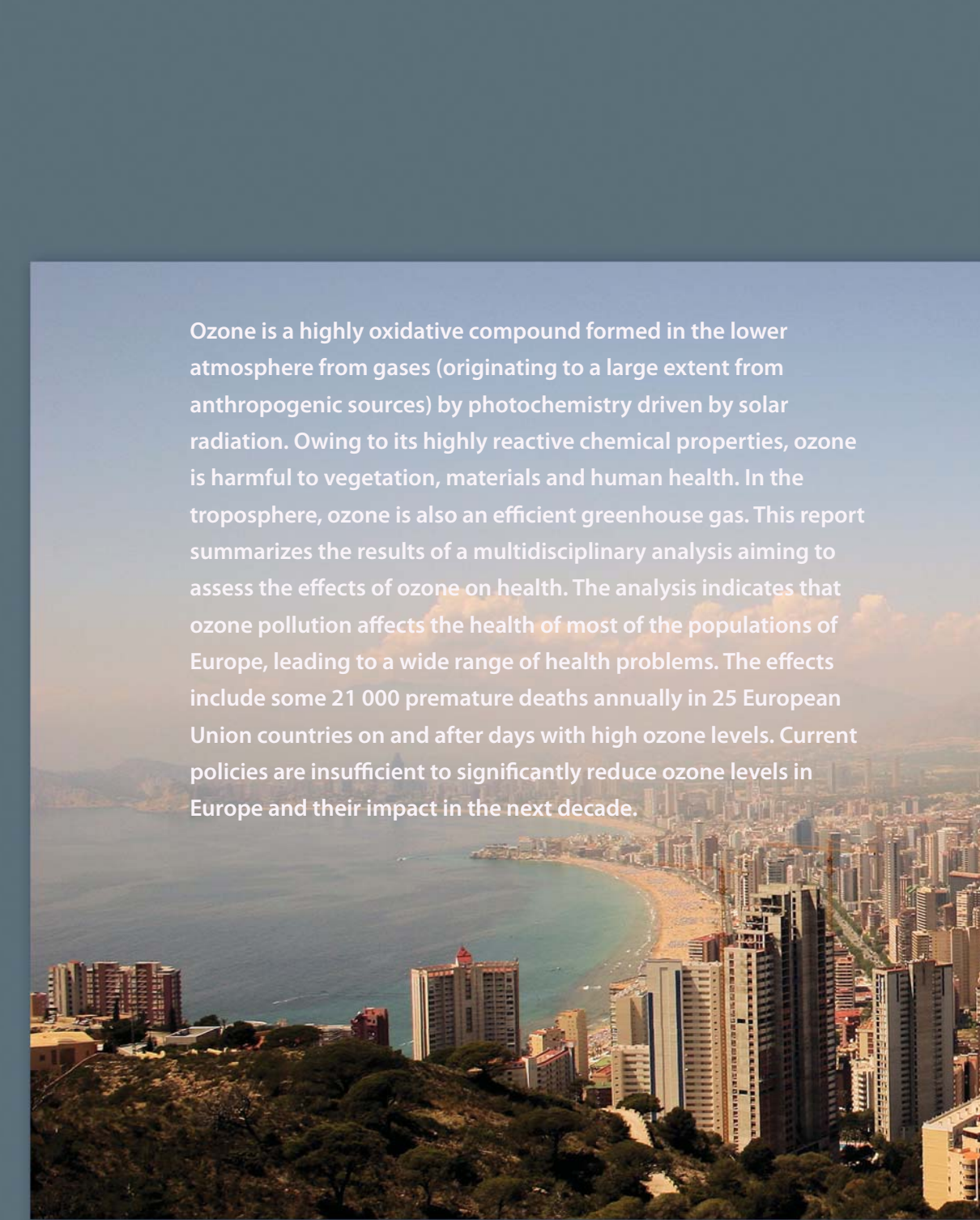
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An aerial photograph of a coastal city, likely Hong Kong, showing a dense urban landscape with numerous high-rise buildings. A prominent feature is a long, curved beach that stretches along the coastline, bordered by the sea on one side and the city on the other. The sky is clear and blue, and the overall scene is captured from an elevated perspective, showing the city's proximity to the water and the surrounding greenery on the hills.

Ozone is a highly oxidative compound formed in the lower atmosphere from gases (originating to a large extent from anthropogenic sources) by photochemistry driven by solar radiation. Owing to its highly reactive chemical properties, ozone is harmful to vegetation, materials and human health. In the troposphere, ozone is also an efficient greenhouse gas. This report summarizes the results of a multidisciplinary analysis aiming to assess the effects of ozone on health. The analysis indicates that ozone pollution affects the health of most of the populations of Europe, leading to a wide range of health problems. The effects include some 21 000 premature deaths annually in 25 European Union countries on and after days with high ozone levels. Current policies are insufficient to significantly reduce ozone levels in Europe and their impact in the next decade.

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